ON THE GENETIC CONSTITUTION OF JERSEY CATTLE, AS INFLUENCED BY INHERITANCE AND ENVIRONMENT[1]

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The high class dairy cow has taken its present form first because of its survival value in nature and second because of centuries of controlled breeding. In both instances the constitution toward which the breeding tended was approached without conscious knowledge of how inheritance was achieved. It was a case of evolution rather than exact breeding based on genetic factors and a known method of inheritance. Yet, after all, this is the way in which man and most of his economically important animals and plants evolved. It is significant to take such a population, analyze it for the mode of inheritance of the characters involved, and amount of heterozygosis which is now extant within the population, determine present distribution of this inheritance, and from this information formulate a picture of the forces which have been at work to guide the animal of today to its present form. The data of this paper and their analyses are presented as a contribution to the quantitative interpretation of this problem as far as it relates to Jersey cattle.

The Island of Jersey lies in the English Channel, but a few miles from the French mainland. There is little doubt but that the remote ancestors of the Jersey breed of cattle were identical with the common stock found on this neighboring mainland, where cattle of similar form and color can still be seen. In the 18th century the cattle of this island, not 30 miles around, were set apart from those of the rest of the world by laws prohibiting the importation of any cattle. These laws have been in force since 1763, or for about 40 generations in the life scale of cattle. The Royal Jersey Agricultural Society has systematically tried to standardize the animals by drawing up scales of points and guiding the breeding operations of the individual farmers. The Jerseys of America were imported from this island stock, most of the importations being from the latter quarter of the 19th century to the present day. The million or so progeny from these imported cattle have been kept separate from the other cattle by registration of pure breeding. They have become distributed over the whole United States.

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A population of 6000 animals scattered over 15 States will obviously vary in heredity and environment, feeding, care, et cetera. If the conformation of these animals is dependent on the variations of inheritance which have been available to such groups, then the parent and offspring and fraternal correlations will furnish a measure of the effect of inheritance. Similarly, if the variations of environment are to be factors in the determination of the size of these animals, then the uniformity of the animals within one herd, as contrasted with those in another, as determined from the proper correlations, measures this effect. But both measures have some part of the other variable within them. Parent and offspring correlations are, in the main, freed from the influences of common environmental variations since years elapse between the growth period of the parent and that of the

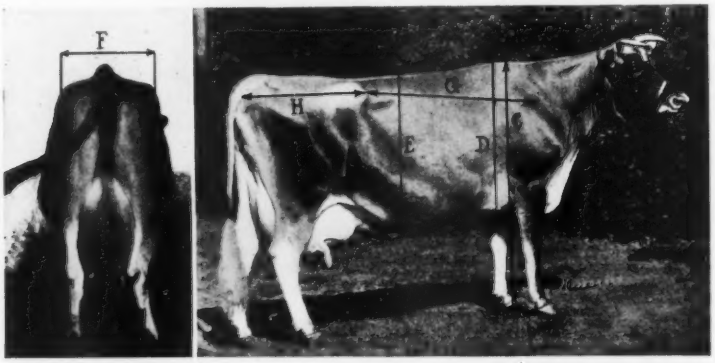

FIGURE 1.—Photographs of Jersey cows showing the points used in making the measurements studied herein.

offspring. This is especially true where the parents and offspring are reared in different herds. The fraternal correlations are not so one-sided a measure of the inheritance as they include also the environmental element of common rearing. The possible influence of selective breeding based on the external appearance of the animal because of its effect on the ancestral correlations enters into this comparison. These questions may be approached through the use of the excellent data on the American Jersey Cattle. (The American Jersey Cattle Club Directors, realizing that the future of any coordinated effort in dairy cattle breeding depends largely on accurate, well analyzed knowledge of all phases of the problem, have had collected the body of information herein described.)

These data include the following measurements on the bull's or cow's type: estimated weight, height at withers, heart girth, paunch girth, width at hips, body length and rump length. Figure 1 shows the relative position

of the measurements taken. About 300 bulls and 6000 cows were measured. The animals come from herds in 15 different States distributed quite generally throughout the United States. Two capable, experienced dairy cattle men took all of the measurements; one man, Mr. RANDOLPH, took all but about 800. The ages, parentages, et cetera of these animals are known through their registration in the herd books. All of the measurements utilized for this study have been age corrected so that all records are strictly comparable for this variable. The volume of material is sufficient to cover the immediate questions: (1) The relation between the sire's conformation and that of his daughters; (2) The correlation between the dam's conformation and that of her daughters; (3) The relation between the types of the full or half sisters; and (4) The assortive mating between sire and dam. (For the study of these data from the viewpoint of conformation and its relation to milk yield et cetera see the literature list (1).)

THE RELATION BETWEEN THE SIRE'S CONFORMATION AND THAT OF HIS DAUGHTERS

For this comparison, there are available 736 pairs of sires and their daughters, some of the pairs being formed by the same sire with different daughters. If the type of the sire is transmitted to the daughter it would be expected that the daughter would resemble the sire's type. By contrasting the measurement of the sire with that of his daughters, it is possible to determine the degree of relationship which exists between their types. The amount of this relationship will be due primarily to the cow's inheritance and secondarily to her environment (such as nutrition, care, et cetera) Table 1 shows the average daughter's size for given conformational measurements of the sires.

The comparison of the sire's type with that of his daughter as shown in table 1 indicated that there is a direct relation between the sire's size and his daughter's conformation. The larger the sire in any given conformational measurement, the larger his daughters tend to be. Thus, sires with a weight of 1025 pounds have daughters whose average weight is 725 pounds, and sires whose weight is 1575 pounds have daughters whose average weight is 1030 pounds. The results, however, are quite irregular, showing that the relation is not a close one. A similar relationship may be drawn for the measurements of sire and daughter in height at withers, depth at withers, heart girth, and paunch girth.

The irregular line of figure 2 represents the actual average daughter's measurements for the given sire's measurements shown in table 1. The straight line shows the general average trend of these measurements. The heavy portion of the line represents the average daughter's measurements which are based on more than 10 individuals.

TABLE 1

Relation between sires' conformations and those of their daughters.

WEIGHT*		HEIGHT AT WITHERS*		DEPTH AT WITHERS		HEART GIRTH		PAUNCH GIRTH		WIDTH AT HIPS		BODY LENGTH		RUMP LENGTH	
SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE	SIRES'	DAUGHTERS' AVERAGE
775	870	47.5	47.0	26.5	26.5	67	70.0	77	83.0	16.5	18.5	31.0	35.7	19.5	19.9
975	685	48.5	46.7	28.5	27.3	71	67.0	79	86.6	17.5	19.2	33.0	36.3	20.5	19.8
1025	725	49.5	47.2	29.5	27.0	73	69.8	81	82.0	18.5	19.4	35.0	36.2	21.5	20.2
1075	970	50.5	47.6	30.5	27.3	75	69.5	83	85.3	19.5	19.7	37.0	—	22.5	20.6
1125	895	51.5	47.3	31.5	27.7	77	70.0	85	87.0	20.5	20.1	39.0	36.1	23.5	21.5
1175	885	52.5	48.3	32.5	27.8	79	70.5	87	85.1	21.5	19.9	41.0	37.0	24.5	21.6
1225	900	53.5	48.2	33.5	27.1	81	72.0	89	87.3	22.5	21.5	43.0	38.4	25.5	21.8
1275	910	54.5	48.1	34.5	27.6	83	71.9	91	87.2	23.5	21.5	45.0	39.2		
1325	895	55.5	49.7	35.5	28.7	85	71.2	93	89.4	24.5	21.1	47.0	40.5		
1375	930	56.5	51.0	37.5	28.8	87	74.2	95	85.8						
1425	925	58.5	48.5			89	70.6	97	88.2						
1475	940							99	89.0						
1525	950							101	89.7						
1575	1030							103	91.6						
1625	970							105	87.9						
1675	1000							107	88.4						
1725	925														

* Weight in pounds, other measurements in inches

These results clearly demonstrate a relation between the size of the sire and the size of his daughter. The relation, however, is not very exact, allowing a fairly large variation in either direction. Two factors in the cow's

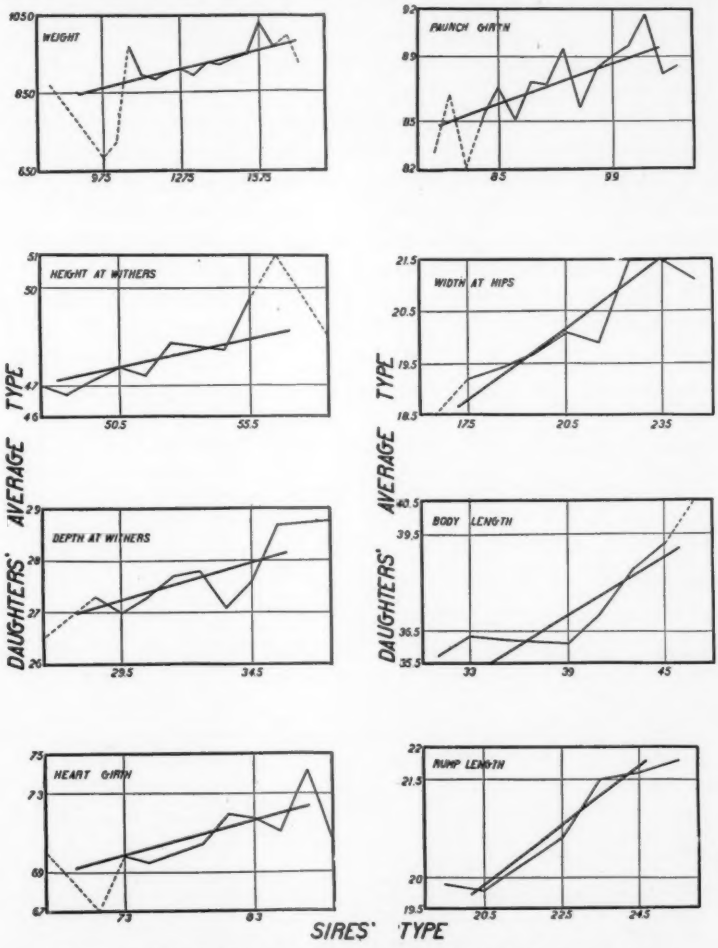

FIGURE 2.—Relation between sire's size and daughter's size in Jersey cattle.

development may bring about such a resemblance between the sire and his daughter: the first is the heredity for size which the sire transmits to his daughter; the second is the condition such as feeding and care which surround each, tending to make them resemble each other. In view of the fact that most sires are grown in other herds and are purchased at an age

when it would be difficult to influence their size, it seems fair to interpret these correlations as due to heredity. The correlations show that the breeder of dairy cattle may reasonably expect his choice of type in the sire to tend to produce daughters which will themselves resemble the sire's type to some degree. As the correlations are quantitatively not high, the residual or uncontrolled variation which would be expected in the type of such daughters would still be large.

If the sire's conformation for one given body measurement, say, height at withers, is compared with the body measurement of the daughter for perhaps body length, it is found that here too there is an intimate relation between the sire's size and the daughter's size. In other words, if the sire selected is of large conformation in a given body part, he tends to beget a daughter of large conformation for other given parts.

The correlation coefficients and prediction equations on which the above conclusions are based are found in table 2.

TABLE 2

Relation between the measurements of the sire and the measurements of his average daughter.

	DAUGHTER'S									
SIRE'S	WEIGHT	HEIGHT AT WITHERS	DEPTH AT WITHERS	HEART GIRTH	PAUNCH GIRTH	WIDTH AT HIPS	BODY LENGTH	RUMP LENGTH	SIRE'S AVERAGE SIZE	STANDARD DEVIATION
Weight	*.34*	.33	.27	.29	.31	.34	.39	.29	1399±11.4	164±8.1
Height at withers	.29	*.31*	.33	.35	.33	.38	.30	.31	52.2± .14	2.0± .10
Depth at withers	.14	.19	*.29*	.28	.38	.38	.34	.32	32.0± .12	1.7± .08
Heart girth	.11	.13	.17	*.30*	.38	.35	.36	.33	80.4± .26	3.7± .18
Paunch girth	.08	.06	.12	.18	*.31*	.32	.34	.24	93.1± .46	6.6± .32
Width at hips	−.01	.07	.09	.32	.32	*.65*	.44	.59	21.1± .12	1.7± .08
Body length	.04	.22	.18	.28	.42	.35	*.42*	.33	41.3± .25	3.0± .17
Rump length	.10	.12	.08	.37	.34	.57	.43	*.51*	22.5± .10	1.4± .07
Daughter's size	933 ±3.0	47.8 ±.04	27.6 ±.03	71.2 ±.09	87.5 ±.14	20.4 ±.03	37.6 ±.07	20.8 ±.03		
Standard deviation	121 ±2.1	1.6 ±.03	1.4 ±.02	3.5 ±.06	5.6 ±.10	1.4 ±.02	2.4 ±.05	1.2 ±.02		

Probable errors: < .06

Sires' and average daughters' conformation

Daughters' weight = 582 + .25 sires' weight
Daughters' height at withers = 34.9 + .25 sires' height at withers
Daughters' depth at withers = 20.0 + .24 sires' depth at withers
Daughters' heart girth = 48.4 + .28 sires' heart girth
Daughters' paunch girth = 63.0 + .26 sires' paunch girth
Daughters' width at hips = 9.1 + .54 sires' width at hips
Daughters' body length = 23.9 + .34 sires' body length
Daughters' rump length = 11.0 + .44 sires' rump length

The correlations on the left to right diagonal line of table 1 are for the measurements alike in sire and daughter. These correlations are in general larger than those where the measurement taken on the sire is different from the measurement taken on the daughter. Such variations result from differential development and from the action of unlike factors in inheritance which affect the body parts separately. Other analytical material is necessary to bring this point into sharp detail.

The block of nine correlations at the extreme lower-right hand corner for the interrelations of width at hips, body length and rump length are distinctly larger than those for the other measurements. This fact will be noted in all of the following tables.

The size of the correlations is also important. Considering like measurements of sire and daughter, the first five measurements have correlations which are somewhat lower than those expected on random assortment of the Mendelian factors on which size may depend. (For a more extended discussion of this question with regard to milk yield and butter fat, see papers cited in reference 2 of the Literature Cited.) The other three are large enough or in one case too large. The quantitative facts, while pointing strongly to the inheritance of these characters along customary multiple factor Mendelian lines, need further amplifications by the data which follow.

INFLUENCE OF DAM'S CONFORMATION ON THAT OF HER DAUGHTERS

The data for the relation between the type of dams and the type of their daughters are more extensive than those of the sires and their daughters, there being practically twice as many daughter and dam pairs. There are 1549 measured daughters which come from measured dams. Furthermore at least half of these daughter-dam pair measurements include different dams, whereas not more than 125 different sires were actually included in the measurements, most of the 736 measurements coming from sires which had more than one daughter. The data on the relation between the dam and daughter are therefore more reliable in this sense than those between sire and daughter. They are more obviously influenced by environment also as the dam and daughter are frequently raised in the same dairy barn. The mean types of the dams and daughters are shown in table 3.

The measurements of table 4 support the conclusion that the dam's conformation predicts the daughter's probable form. The results, however, are fairly irregular, indicating that the relation is not a close one. The data of table 3 are presented in graphical form in figure 3.

The irregular line in figure 3 shows the actual average daughter's size for a given conformation of the dam. The heavy line shows those points based on more than 10 daughters in determining the average. The straight line

TABLE 3

Relation between dam's type and that of her daughter.

WEIGHT		HEIGHT AT WITHERS		DEPTH AT WITHERS		HEART GIRTH		PAUNCH GIRTH		WIDTH AT HIPS		BODY LENGTH		RUMP LENGTH	
DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE	DAMS'	DAUGHTERS' AVERAGE
425	1125	42.5	46.3	20.5	27.5	61	67.3	67	91.0	16.5	18.2	29.5	38.5	16.5	20.5
575	975	43.5	46.2	21.5	27.5	63	68.4	69		17.5	18.8	30.5		17.5	18.8
625	865	44.5	46.5	22.5	26.0	65	67.0	71	79.6	18.5	19.1	31.5	36.0	18.5	18.9
675	920	45.5	46.9	23.5	26.2	67	68.7	73	83.0	19.5	19.6	32.5	34.9	19.5	19.8
725	806	46.5	47.1	24.5	26.7	69	69.7	75	98.5	20.5	20.1	33.5	35.1	20.5	20.4
775	835	47.5	47.5	25.5	26.7	71	70.7	77	82.2	21.5	21.1	34.5	35.3	21.5	21.3
825	875	48.5	48.1	26.5	27.2	73	69.8	79	83.8	22.5	21.6	35.5	36.3	22.5	21.7
875	885	49.5	48.4	27.5	27.0	75	70.4	81	84.6	23.5	21.8	36.5	36.6	23.5	22.4
925	900	50.5	48.9	28.5	27.9	77	72.8	83	85.4	24.5	21.9	37.5	37.2	24.5	22.1
975	932	51.5	45.6	29.5	28.0	79	74.0	85	85.8	25.5		38.5	38.1		
1025	963	52.5	48.8	30.5	28.4	81	74.7	87	86.8	26.5		39.5	38.6		
1075	975			31.5	28.3	83	75.0	89	88.0	27.5	17.5	40.5	38.6		
1125	995			32.5	29.0			91	89.2	28.5	18.5	41.5	39.7		
1175	1010							93	88.6			42.5	40.3		
1225	1005							95	89.4			43.5	38.3		
1275	1025							97	89.8			44.5	39.5		
								99	90.6			45.5	37.5		
								101	94.1			46.5	38.0		
								103							

TABLE 4

Relation between the conformation of the daughter and that of her dam.

DAMS' MEASUREMENTS	DAUGHTERS' MEASUREMENTS									
	WEIGHT	HEIGHT AT WITHERS	DEPTH AT WITHERS	HEART GIRTH	PAUNCH GIRTH	WIDTH AT HIPS	BODY LENGTH	RUMP LENGTH	DAMS' AVERAGE SIZE	STANDARD DEVIATION
Weight	*.37*	.32	.26	.26	.24	.15	.18	.20	917±1.9	113±1.4
Height at withers	.25	*.40*	.27	.25	.16	.19	.19	.25	47.6± .03	1.7± .02
Depth at withers	.21	.31	*.32*	.35	.27	.34	.30	.38	27.5± .02	1.5± .02
Heart girth	.18	.27	.32	*.42*	.34	.43	.34	.44	71.1± .06	3.5± .04
Paunch girth	.15	.23	.28	.37	*.35*	.36	.33	.38	87.5± .10	5.4± .06
Width at hips	.10	.21	.26	.40	.33	*.59*	.50	.58	20.3± .02	1.6± .02
Body length	.16	.29	.33	.36	.29	.49	*.49*	.46	37.1± .05	2.4± .03
Rump length	.11	.25	.31	.43	.34	.58	.49	*.64*	20.5± .02	1.4± .02
Daughters' average size	905± 2.1	47.6± .03	27.5± .02	70.7± .06	87.0± .10	20.2± .02	37.1± .05	20.5± .02		
Standard deviation	120± 1.5	1.6± .02	1.4± .02	3.7± .04	5.7± .07	1.5± .02	2.3± .03	1.3± .02		

Probable errors 0.02 when r less than 0.3, otherwise the probable errors are 0.01.

Daughters' weight = 540 + .398 Dams' weight
Daughters' height at withers = 28.7 + .398 Dams' height at withers
Daughters' depth at withers = 18.9 + .312 Dams' depth at withers
Daughters' heart girth = 38.7 + .450 Dams' heart girth
Daughters' paunch girth = 54.4 + .373 Dams' paunch girth
Daughters' width at hips = 8.9 + .555 Dams' width at hips
Daughters' body length = 19.3 + .481 Dams' body length
Daughters' rump length = 8.0 + .612 Dams' rump length

indicates the average weighted trend of the data as determined from the proper equations indicated in table 4.

The size of the daughters of a small Jersey cow compared with the size of

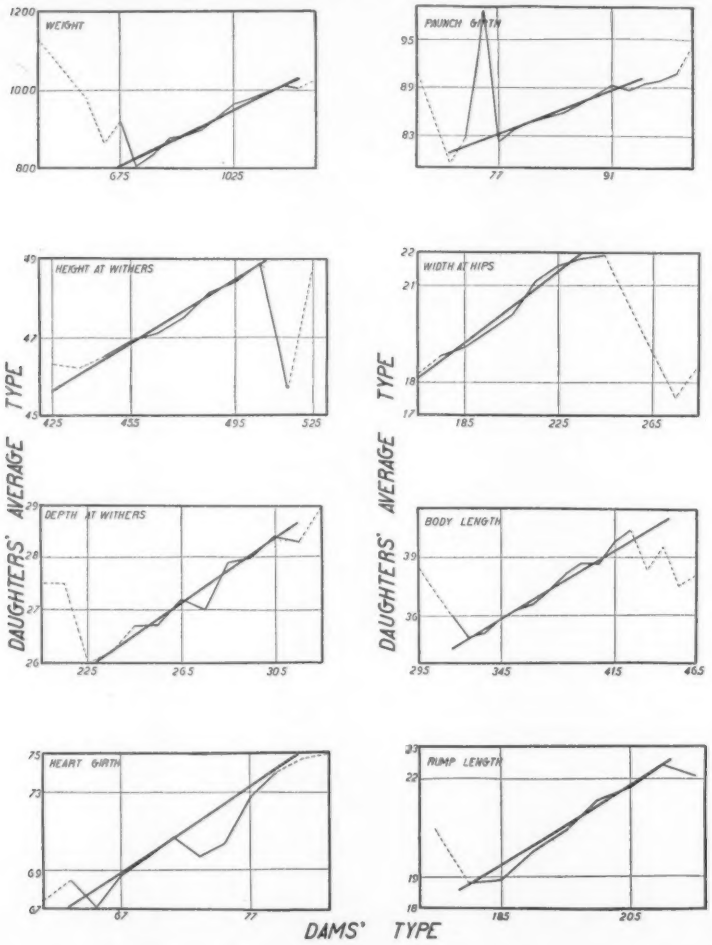

FIGURE 3.—The relation between the conformation of the dam and the average conformation of her daughter.

the daughters of a large Jersey cow indicates what size may mean to the offspring when considered on the basis of the extremes of the race. If the dam is 575 pounds, the average size of the daughter is 769 pounds. If the

dam is 1225 pounds, the average size of the daughter is 1028 pounds. An increase in size of 650 pounds for the dam has resulted in an average increase in size for the daughters of 259 pounds. For height at withers dams of 41.5 inches have, on the average, daughters of 45.2 inches in size. Where the dam is 51.5 inches at the withers, the average daughter's size is 49.2 inches. It is clear that the daughters tend to approach the average although they still show the effect of the dams from which they come. An increase of 10 inches for the size of the dam at height at withers has resulted in an increase of 4 inches for the daughter. A similar condition is found for the other body measurements.

The correlation coefficients and the proper equations for relating the size of the dam to that of the daughter are given in table 4.

The data of table 4 are comparable with those of table 2 for the sire. The average correlation coefficient between the daughters and sires on like measurements is 0.39 and that for the dams and daughters is 0.45. These correlations are not significantly different although they indicate a slight influence of the more common environment of the dam and daughter. If we are to interpret these relationships as due to the inheritance of size on a multiple factor basis the numerical value is somewhat low. The interrelation of the unlike body parts shows somewhat the same tendency. The mean correlation coefficient for the sires is 0.27 and that for the dams is 0.30.

RELATION BETWEEN THE CONFORMATIONS OF FULL SISTERS

Full sisters are, from an inheritance standpoint, as closely related individuals as it is possible to have in only one generation of matings. It would be expected that full sisters would resemble each other closely if inheritance in any way played a part in determining the conformation of the animal. It would also be expected, in view of the fact that most full sisters are brought up under the same conditions, in the same barn, by the same owner, that environment too would be an important factor in bringing about a close resemblance between such relatives. The effect of environment would undoubtedly be much greater for such full sisters than for either a sire and his daughter or a dam and her daughter. The results of the comparison of full sisters are important in showing the combined influence of these two factors in molding the conformation of the animal to the desired type. The measurements on 292 full sisters, making a total of 802 pairs, have been compared for the eight items of conformation.

The results of this study show that the conformations of full sisters are more closely related in the different measurements than are either sire and daughter or dam and daughter. The correlation coefficients showing the degree of these relationships are given in table 5. The close relation which

TABLE 5

Relation between the conformation of one sister with that of her full sister.

FULL SISTER'S MEASUREMENTS	SISTER'S MEASUREMENTS	CORRELATION COEFFICIENTS	MEAN	STANDARD DEVIATION
Weight	Weight	.49	936 ±3.3	125±2.3
Height at withers	Height at withers	.43	48.0± .05	1.66± .03
Depth at withers	Depth at withers	.33	27.7± .05	1.38± .03
Heart girth	Heart girth	.40	71.6± .10	3.57± .07
Paunch girth	Paunch girth	.37	88.2± .14	5.08± .10
Width at hips	Width at hips	.65	20.5± .04	1.44± .03
Body length	Body length	.54	37.6± .12	2.53± .08
Rump length	Rump length	.64	20.7± .04	1.29± .02

Full sister's weight =475.2+.49 sister's weight
Full sister's height at withers= 27.5+.43 sister's height at withers
Full sister's depth at withers = 18.5+.33 sister's depth at withers
Full sister's heart girth = 43.0+.40 sister's heart girth
Full sister's paunch girth = 55.2+.37 sister's paunch girth
Full sister's width at hips = 7.1+.65 sister's width at hips
Full sister's body length = 17.4+.54 sister's body length
Full sister's rump length = 7.4+.64 sister's rump length

exists between these full sisters may be indicated in the following way. If the full sisters of cows weighing 800 pounds are compared with the full sisters of cows weighing 900 pounds, the difference in the average weight of these full sister groups would furnish an indication of the influence of differences of 100 pounds of weight on the weight of another full sister. By such a comparison, it is found that an increase in any of the type measurements results in an increase in the type measurements of the second full sisters as follows: A cow which weighs 100 pounds more than another cow tends to have full sisters 49 pounds heavier than those of the smaller cow. Similarly, for cows differing by one inch in size, the larger cow tends to have full sisters 0.42 inches larger in height at withers, 0.34 inches in depth at withers, 0.40 inches in heart girth, 0.37 inches in paunch girth, 0.65 inches in width at hips, 0.54 inches in body length, and 0.64 inches in rump length. It will be noted that the rates of increase are essentially the same for the four measurements, height at withers, depth at withers, heart girth, and paunch girth. Those for width at hips, body length, and rump length are considerably greater. The results are throughout concordant in showing the marked influence of the cow's type on that of her full sister.

The correlation coefficients measuring the relation between full sisters, together with the equations showing the mean values for the measurements, are found in table 5. The average correlation is 0.48, a correlation which is quite similar to that expected for full sisters on random mating and complete determination by heredity. In view of the divergence of the first five correlations from the values of the last three, this relation has little meaning without the other data which are to follow.

RELATION BETWEEN THE TYPE OF ONE HALF SISTER WITH THAT OF ANOTHER WHEN THE SIRE IS THE COMMON PARENT

Half sisters may have a common sire or a common dam. On an inheritance basis they should have approximately only half the inheritance in common which is found for full sisters.

On the other hand, half sisters have the same opportunity with full sisters to be affected by the different feeding, management and selection practices of the various farms from which they come. Most half sister groups come from the same farms rather than different farms and would therefore tend to be alike both because of their inheritance and their environment. Taking these factors into account, the degree of resemblance between full sisters on the one hand would be in its simplest form:

$$1/2\ \text{heredity}^2+\text{environment}^2$$

while for half sisters:

$$1/4\ \text{heredity}^2+\text{environment}^2$$

The expectation would consequently be that the half sisters would resemble each other less than full sisters in their size measurements; the differences being interpreted as the effects of the environment. If selection and care, et cetera, have no effect, then the half sisters should resemble each other only half as much as the full sisters. If environment plays an important role, the half sisters will be nearly as much alike as the full sisters. If heredity has no effect the half sisters will be as much alike as full sisters. The full sister groups and half sister groups, coming as they do from all manner of farms each of which have their chance to influence the conformation of pairs of sisters, give the environmental factors full play and push to the background the influence of heredity. Any explanation of the results must take cognizance of the influence of the two components of heredity and environment so that the facts may be truly comparable with those previously obtained in the parent and offspring groups.

If the cow's conformation be contrasted with that of her half sister, on the same basis as that used for the full sisters, it is found that for an increase of 100 pounds in weight the larger cow tends to have half sisters 38 pounds heavier than the smaller cow. An increase of one inch in any of the body measurements results in the larger cow having half sisters larger by the following amounts: height at withers 0.34 inches, depth at withers 0.28 inches, heart girth 0.37 inches, paunch girth 0.31 inches, width at hips 0.67 inches, body length 0.60 inches, and rump length 0.50 inches. It is clear that the half sisters do not resemble each other to quite the same extent as full sisters do. It is also noted that the first five measurements tend to have the same relative effect, whereas the last three tend to have a much greater effect, so far as the influence of the size of one half sister

on that of the other is concerned. Some factor, not as yet clear, tends to make the cows resemble each other more closely in width at hips, body length, and rump length than they do for the other five measurements, weight, height at withers, depth at withers, heart girth, and paunch girth.

The correlations measuring these relationships are shown in table 6.

TABLE 6

Relation between the conformation of one sister with that of her half sister on the sire's side.

HALF SISTER'S MEASUREMENTS	SISTER'S MEASUREMENTS	CORRELATION COEFFICIENTS	MEAN	STANDARD DEVIATION
Weight	Weight	.38	920±1.3	116± .9
Height at withers	Height at withers	.34	47.7± .02	1.63± .01
Depth at withers	Depth at withers	.28	27.6± .02	1.45± .01
Heart girth	Heart girth	.37	71.4± .04	3.71± .03
Paunch girth	Paunch girth	.31	87.8± .06	5.48± .04
Width at hips	Width at hips	.67	20.6± .02	1.51± .01
Body length	Body length	.50	37.7± .03	2.44± .02
Rump length	Rump length	.60	20.9± .02	1.38± .01

Half sister's weight = 574 + .38 sister's weight
Half sister's height at withers = 31.5 + .34 sister's height at withers
Half sister's depth at withers = 19.8 + .28 sister's depth at withers
Half sister's heart girth = 44.8 + .37 sister's heart girth
Half sister's paunch girth = 60.7 + .31 sister's paunch girth
Half sister's width at hips = 6.8 + .67 sister's width at hips
Half sister's body length = 18.8 + .50 sister's body length
Half sister's rump length = 8.4 + .60 sister's rump length

RELATION BETWEEN THE CONFORMATION OF ONE HALF SISTER WITH THAT OF ANOTHER WHEN THE DAM IS THE COMMON PARENT

Half sisters with a common dam have the same common inheritance as half sisters with a common sire.

If the influence of one half sister upon another be determined in the manner previously used, it is found that a cow which is 100 pounds heavier than another will tend to have half sisters 39 pounds heavier than those of the smaller cow. Similarly the cow which is larger by one inch for the other body measurements will have sisters larger by the following amounts: 0.32 for height at withers; 0.26 for depth at withers; 0.42 for heart girth; 0.38 for paunch girth; 0.55 for width at hips; 0.48 for body length; and 0.60 for rump length. These results show clearly that the size of the cow materially affects the size of her half sisters. This result is again most pronounced for width at hips, body length, and rump length, but is significant for weight, height at withers, depth at withers, paunch girth, and heart girth.

The correlation coefficients of table 7 for the interrelation of these half sisters' measurements have a mean value of 0.42. This value is identical with that of the half sisters on the sire's side and somewhat less than that for the full sisters.

TABLE 7

Relation between the conformation of one sister with that of her half sister on the dam's side.

HALF SISTER'S MEASUREMENTS	SISTER'S MEASUREMENTS	CORRELATION COEFFICIENTS	MEAN	STANDARD DEVIATION
Weight	Weight	.39	917±2.7	117±1.9
Height at withers	Height at withers	.32	47.7± .04	1.66± .03
Depth at withers	Depth at withers	.26	27.5± .03	1.44± .02
Hearth girth	Heart girth	.42	71.1± .08	3.53± .06
Paunch girth	Paunch girth	.38	87.5± .13	5.66± .09
Width at hips	Width at hips	.55	20.3± .03	1.46± .02
Body length	Body length	.48	37.1± .06	2.33± .04
Rump length	Rump length	.60	20.5± .03	1.29± .02

Half sister's weight = 557 + .39 sister's weight
Half sister's height at withers = 32.2 + .32 sister's height at withers
Half sister's depth at withers = 20.3 + .26 sister's depth at withers
Half sister's heart girth = 41.5 + .42 sister's heart girth
Half sister's paunch girth = 54.3 + .38 sister's paunch girth
Half sister's width at hips = 9.2 + .55 sister's width at hips
Half sister's body length = 19.3 + .48 sister's body length
Half sister's rump length = 8.2 + .60 sister's rump length

ASSORTIVE MATING IN JERSEY CATTLE

By assortive mating is meant the tendency of breeders to mate individuals which are alike. This similarity may be taken as a whole or it may be for some given part of the body. Assortive mating is thus a distinct and fundamental function in any breeding, whether the procedure be deliberately controlled or not.

In regard to one character, milk yield, it was found that assortive mating did not occur to the extent which might be expected. This was no doubt due in large part to the fact that milk yield is a relatively obscure character requiring a large volume of records for its appreciation and control, a fact tending to reduce the number of those willing to give it sufficient attention to make for an effective assortive mating coefficient. The case for the body characteristics of the dairy cow is not of this kind. The relative size of any of the characters may be readily appreciated by simply looking at the cow. Dairy-men are continually making comparisons of this kind and naturally become expert in taking into account such things as relation of size to age, thus making the selection more effective.

Assortive mating coefficients thus have an interest to all breeders because they measure the effective selection and breeding which is taking

place within the breed for the given character. They have a further interest in that the degree to which the parents are selected for a given character markedly influences the amount of resemblance which will be found between the parents and their offspring.

If assortive mating is to be a real force in molding the characteristics of a breed it must do several things. The first and most important is that the sires to be used in the different herds must, in large part, come from superior parents. The sires selected for actual breeding must themselves be the best of the progeny of such parents. Sires of this calibre should then have the best mates which the breed affords. Some appreciation of how active is this selective breeding may be had by a comparison of the relative sizes of the sires and the cows to which they are bred. The results of this comparison for the eight body measurements of conformation are shown in the averages which follow; the sires being divided into groups, from the smallest to the largest, three groups in all and their mates' average sizes determined.

WEIGHT		HEIGHT AT WITHERS		DEPTH AT WITHERS		HEART GIRTH	
SIRE	MATE	SIRE	MATE	SIRE	MATE	SIRE	MATE
1199	880	49.9	47.4	29.7	27.3	74.3	70.0
1430	925	52.0	48.0	32.1	27.5	80.0	71.0
1570	975	54.5	47.6	34.8	27.5	84.2	72.0

PAUNCH GIRTH		WIDTH AT HIPS		BODY LENGTH		RUMP LENGTH	
SIRE	MATE	SIRE	MATE	SIRE	MATE	SIRE	MATE
84.0	85.6	19.1	19.5	37.1	36.2	21.2	19.9
92.0	88.6	20.8	20.2	41.1	36.8	22.5	20.6
99.1	88.0	23.0	22.0	43.5	38.5	24.3	21.8

It will be recalled that all measurements presented are mature form measurements. The data cited above were obtained in the following manner. There were 103 matings of sires and dams in which the sire's weight was less than 1350 pounds. These sires' weights averaged 1199 pounds. They were bred to cows whose average weight was 880 pounds. There were 167 matings in which the sire's weight ranged between 1350 and 1500 pounds with an average of 1430 pounds. They were bred to cows averaging 925 pounds. Seventy-seven matings had sires whose weights were above 1500 pounds. These sires averaged 1570 pounds in weight and were bred to cows averaging 975 pounds in weight. There is a distinct increase in the weight of the sires' mates as the weight of the sires increases. This correlation is carried through the other data particularly for the width at hips, body length and rump length. These results may be seen graphically in figure 4.

The degree of selection practiced in the Jerseys is evidently pronounced for some of the regions of the body conformation, less in others and almost non-existent in the remainder. The regions chosen for emphasis in breeding are width at the hips, body length and rump length. Less effective selection

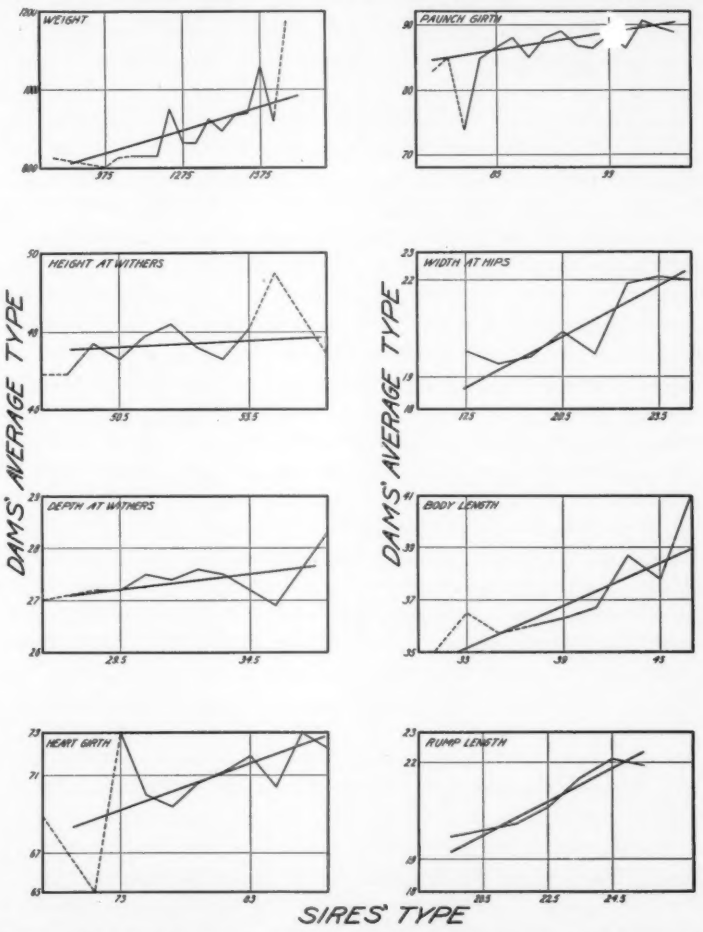

FIGURE 4.—Relation between the size of the sire and the size of his mates for Jersey cattle.

is seen in body weight, heart girth and paunch girth. Little selection is practiced for height at withers and depth at withers. Herd owners very evidently believe that if they have cows which have wide hips, long bodies or rumps, that these cows should be bred to bulls having the same qualities,

whereas if their cows have narrow hips, short bodies or rumps, they should be bred to bulls which, relatively speaking, have equally narrow hips, short bodies and rumps. A like, though less extreme, belief also appears to exist with regard to the weight, heart girth and paunch girth. The matings with regard to the height at withers and depth at withers appear to be almost random matings, a sire high at the withers being proportionately as often bred to cows which were low at the withers, high at the withers, or medium in height.

Two factors appear to be capable of giving the degree of assortive mating observed. The first is that breeders may believe that the conformational characteristics, width at hips, rump length and body length, and, to a lesser degree, weight, paunch girth and heart girth in the order named are factors of importance in indicating production. If such a belief is sufficiently strong to cause these points to have a financial premium the desired sires and dams would be concentrated into the hands of the wealthy, the less desired stock would be in the herds of breeders with less wealth, thus giving the observed results. This situation could be accentuated by a second factor working in conjunction with it. This second factor is the tendency, perhaps stronger in the Jersey breed than in most others, of confining the breeding closely within family lines. If these family lines differ by characteristics such as width at hips, body length, et cetera, these characteristics being inherited and selected according to the particular whim of the breeders establishing the family, they would tend to make diverse lines for these characteristics. A population composed of such distinct families would give the assortive mating coefficients like those observed. In passing it is of point to suggest that it is unfortunate that the points of conformation most important to production were not emphasized in this selection: weight and heart girth.

The relative intensity of the selective or assortive breeding going on within the herds of Jerseys in this country is best appreciated by an examination of the correlation coefficients between the measurements of sires and their mates. These correlation coefficients are given in the tables which follow.

TABLE 8

Correlation coefficients between the sire's measurements and those of his mates.

	CORRELATION COEFFICIENTS
Weight	0.26 ± .03
Height at Withers	.08 ± .04
Depth at Withers	.07 ± .04
Hearth Girth	.18 ± .03
Paunch Girth	.22 ± .03
Width at Hips	.65 ± .02
Body Length	.40 ± .03
Rump Length	.52 ± .03

The first five assortive mating coefficients of table 8 correspond well with those which PEARSON (3) has calculated for stature, span and forearm length in man. The last three coefficients differ from either set in being much larger. The size of these coefficients suggests that they are in part responsible for the observed difference in the ancestral correlations of these same groups. The analysis of this point is presented in the following section.

RELATIVE INFLUENCE OF HEREDITY AND ENVIRONMENT ON CONFORMATION WITHIN THESE JERSEYS

The somatic appearance of an animal is due, first, to the genetic constitution of the fertilized egg and, second, to the environmental opportunity which is afforded the egg for development up to adult size. Assortive mating based on somatic appearance of the animal would consequently be attributed to both heredity and environment.

The definite tendency of Jersey breeders to breed within families would give the assortive mating coefficients a different emphasis. The families within the Jerseys are genetically different in origin. The average environment as represented by care, climate, feeding, et cetera, is throughout essentially the same between the different families. There are good, bad and indifferent cattlemen growing cattle in each family. The magnitude of the assortive mating coefficient when the breeding is carried on within such sub-groups is consequently dependent on their genetic background.

These two interpretations appear to be the major possibilities in analyzing the coefficients of assortive mating. There is another possibility, however; namely, that in taking the measurements the standard of measurement was different between the men making the measurements. This possibility can be eliminated since the correlations for the data of the two men derived separately lead to essentially the same numerical results. To remove any possible objection on this ground we shall consider only the data taken by one man, measuring the larger series of about 5000 animals.

As WRIGHT, FISHER and others (4, 5) have pointed out, the parent and offspring correlations are less than the full sister correlations by an amount equal to the effect of the common environment or, as FISHER (6) has shown to the influence of dominance. In our case any difference between the sire and daughter correlations and the dam and daughter correlations might be considered due to the more common environment of dam and daughter in their development. In his systems of mating, WRIGHT[2] has analyzed these assumptions and derived general formulae for them. His symbols are h^2 determination of size by heredity, d^2 determination of heredity by dominance deviations, e^2 determination of size by the environment com-

[2] The writer is indebted to Dr. WRIGHT for much in the discussion which follows.

mon to full or half sisters, m the correlation between genetic constitution of mates, and m_1 correlation between genotypes of different mates. The effects of the common environment and of dominance could theoretically be combined in all gradations. Such gradation would offer serious complication in the analysis of the problem. An estimate of the effect of common environment and dominance can be reached, however, (1) by assuming the dominance effect to be non-existent, and (2) by assuming the environmental effect to be absent and the dominance to account for the differences. By contrasting the two results it is then possible to see what effect each of the variables really has. The equations which follow on the various assumptions are listed below:

(1) Assortive mating is somatic and the genetic factors are without dominance.

$$r_{sd}=\frac{m}{h^2} \qquad r_{so}=r_{do}=1/2h^2(1+r_{sd})$$

$$r_{oo}=1/2h^2(1+m)+e^2 \qquad r_{oo}'=1/4h^2(1+2m+m^1)+e_1^2$$

Where s = sire, d dam, o daughter, oo full sister and oo^1 half sister.

(2) Assortive mating as above, but difference between dam and daughter correlations and sire and daughter correlations assumed due to the more common environment of dam and daughter as contrasted with that of sire and daughter. The only change which this assumption makes in the above equations is

$r_{sd}=1/2h^2(1+r_{sd})$ as before but $r_{do}=1/2h^2(1+r_{sd})+e^2$

(3) Assortive mating as above but with genetic factors showing dominance and no effect of environment.

$$r_{sd}=\frac{m}{h^2(1-d^2)} \qquad r_{so}=r_{do}=1/2h^2(1-d^2)(1+r_{sd})$$

$$r_{oo}=1/2h^2(1-\frac{d^2}{2})(1+m) \qquad r_{oo}'=1/4h^2(1-d^2)(1+2m+m^1)$$

(4) Assortive mating is genetic and no dominance of factors, environment as before.

$$r_{sd}=h^2m \qquad r_{so}=r_{do}=1/2h^2(1+m)$$

$$r_{oo}=1/2h^2(1+m)+e_1^2 \qquad r_{oo}'=1/4h^2(1+2m+m^1)+e_1^2$$

The correlation coefficients which are derived from the series of measurements taken by the man measuring the largest series are shown below. These correlations will be noted to be entirely similar to those derived from the combined data of both men. There was consequently no divergence of the standard used by the two men in measuring their animals.

TABLE 9

	r_{so}	r_{do}	r_{sd}	r_{co}	$r_{oo'}$ sire	$r_{oo'}$ dam
Weight	0.349	0.382	0.212	0.493	0.353	0.291
Height at Withers	0.267	0.336	0.013	0.383	0.306	0.223
Depth at Withers	0.359	0.312	0.108	0.304	0.283	0.219
Heart Girth	0.350	0.436	0.218	0.450	0.381	0.416
Paunch Girth	0.299	0.376	0.204	0.383	0.320	0.377
Width at Hips	0.686	0.622	0.607	0.688	0.614	0.579
Body Length	0.407	0.500	0.326	0.492	0.505	0.513
Rump Length	0.569	0.679	0.571	0.705	0.615	0.629

The solution of the various equations under the above assumptions is shown below.

The data of table 10 enable us to form some judgment regarding the influence of the various factors which affect size inheritance. The hypotheses considered are the major simple ones. Theoretically they could be combined in a variety of ways. The results from such combinations would be intermediate, however, so that conclusions arrived at on the basis of this material would be essentially correct for such intermediate hypotheses.

The data under hypotheses (1) and (2) with assortive mating somatic and no dominance of the genetic factors for size and that for (4) with assortive mating genetic and no dominance of these same factors, give the factor environment all the possible credit it can have in determining the constitution of these Jersey cattle. The data show that the environmental effects are negligible. In fact it will be noted that several of the constants take negative values. The facts in these dairy cattle consequently agree with those of PEARSON and LEE (3) on human constitution in showing but little effect on the constitution of the environmental variation which differentially played upon the individuals within the populations studied.

The hypothecating of dominance in the factors for size as seen in (3) leads to high values for the effect of heredity; three values being in fact indeterminate. The values for the correlation between the genotypes of mates are also high, four out of the eight being indeterminate. These facts seem to give suitable grounds for discarding the dominance hypothesis for the size factors in these dairy cattle.

Further information by which to differentiate between the effectiveness of the other hypotheses is furnished by the genetic correlations for the ancestral combinations as calculated from these data.

The consequences following from these three hypotheses as seen in tables 10 and 11 certainly suggest that (4), assortive mating of genetic origin and no dominance is not applicable to these Jersey cattle. The genetic correlations are throughout too high. Such correlations would require the Jerseys to be split into families which were only bred within each other and never

TABLE 10

Relative influence of heredity h^2, environment e^2, dominance d^2, and the correlation of genotypes m, m^1.

	(1)				(2)					(3)				(4)			
	h^2	e^2	m	m^1	h^2	e_1^2	e_2^2	m	m^1	h^2	d^2	m	m^1	h^2	e^2	m	m^1
Weight	0.60	0.15	0.13	−0.14	0.58	0.17	0.03	0.12	−0.19	1.22	0.50	0.13	0.88	0.52	0.13	0.41	0.32
Height at Withers	0.60	0.06	0.08	0.21	0.53	0.10	0.07	0.07	0.10	0.85	0.30	0.08	0.62	0.59	0.08	0.02	0.20
Depth at Withers	0.61	−0.02	0.07	0.41	0.65	−0.04	−0.05	0.07	0.67	0.68	0.12	0.07	0.53	0.56	−0.03	0.19	0.18
Heart Girth	0.65	0.08	0.14	0.68	0.58	0.13	0.00	0.13	0.64	0.98	0.34	0.14	1.19	0.57	0.06	0.38	0.63
Paunch Girth	0.56	0.07	0.11	0.75	0.50	0.11	0.08	0.10	0.72	0.85	0.34	0.11	1.26	0.47	0.05	0.43	0.70
Width at Hips	0.81	0.08	0.50	0.55	0.85	0.04	−0.06	0.52	0.57	1.13	0.28	0.50	0.94	0.70	0.03	0.87	0.43
Body Length	0.68	0.07	0.22	1.10	0.61	0.12	0.09	0.20	1.11	0.98	0.30	0.22	1.53	0.58	0.04	0.56	1.12
Rump Length	0.79	0.13	0.45	0.59	0.72	0.19	0.11	0.41	0.55	1.30	0.39	0.45	1.23	0.68	0.08	0.84	0.51
Average	0.66	0.08	0.21		0.63	0.11		0.30			0.32	0.21		0.58		0.46	
	±.02	±.01	±.04		±.03	±.01		±.04			±.02	±.04		±.02		±.06	

outside. This type of breeding is partly true but inbreeding should be widespread if it were as active as the correlations under (4) would necessitate.

TABLE 11

Genetic correlations.

	(1)			(2)			(4)		
	r_{po}	r_{oo}	$r_{oo'}$	r_{po}	r_{oo}	$r_{oo'}$	r_{po}	r_{oo}	$r_{oo'}$
Weight	0.61	0.56	0.28	0.61	0.56	0.26	0.70	0.70	0.53
Height at Withers	0.51	0.54	0.34	0.51	0.53	0.31	0.51	0.51	0.31
Depth at Withers	0.55	0.53	0.39	0.55	0.54	0.45	0.60	0.60	0.39
Heart Girth	0.61	0.57	0.49	0.61	0.56	0.47	0.69	0.69	0.60
Paunch Girth	0.60	0.56	0.50	0.60	0.55	0.48	0.72	0.72	0.64
Width at Hips	0.80	0.75	0.64	0.80	0.76	0.65	0.93	0.93	0.80
Body Length	0.66	0.61	0.64	0.66	0.66	0.63	0.78	0.78	0.81
Rump Length	0.79	0.73	0.62	0.79	0.71	0.59	0.92	0.92	0.80

Hypotheses 1 and 2 are essentially the same in view of the lack of any environmental effect. The results are, considering their probable errors, reasonable although the variation covered is quite wide. The greatest complicating factor is the half sister correlations which are too large. The best hypothesis would seem to be that part of the assortive mating is somatic and part genetic, the factors for size being without dominance and mating taking place in such a manner as to form partially non-interbreeding families.

That variation in heredity is the main cause of the variation in the size of Jersey cattle is evident from this analysis. The close relation between the genotypes of the mates has resulted from the type of breeding. It is of interest to note how far this inbreeding and selection has been carried by the herd owners within their individual herds and how much residual variation

TABLE 12

Correlation coefficients for the relation of dam and daughter type within certain herds.

HERD NO.	WEIGHT	HEIGHT AT WITHERS	DEPTH AT WITHERS	HEART GIRTH	PAUNCH GIRTH	WIDTH AT HIPS	BODY LENGTH	RUMP LENGTH	NO. OF PAIRS
19	−0.22	−0.13	−0.04	−0.01	0.03	−0.01	0.15	0.02	27
27	−0.03	0.10	−0.32	−0.11	−0.20	0.08	0.31	0.20	25
47	0.06	−0.38	0.26	0.09	−0.31	0.08	0.17	0.10	23
52	0.45	0.34	0.27	0.43	0.28	0.32	0.16	0.39	20
58	0.53	0.49	0.65	0.32	0.49	0.34	0.18	0.60	27
69	0.30	0.26	0.18	0.17	0.35	0.51	0.35	0.16	27
77	−0.15	0.23	−0.13	−0.12	−0.02	−0.15	−0.15	0.15	50
78	0.06	0.25	0.01	−0.02	−0.02	−0.02	−0.12	0.04	63
88	0.33	0.34	0.44	0.30	0.31	0.15	0.15	0.21	27
102	0.03	0.20	0.18	0.05	−0.04	0.04	0.26	0.13	39
203	−0.02	0.39	0.20	0.12	−0.05	−0.08	0.07	0.34	31
Mean	0.12	0.19	0.15	0.11	0.07	0.11	0.13	0.20	

still exists which the individual breeder may utilize in further change of the constitution of his cows. This information may be derived from the correlations of conformations of cows within the individual herds. Because of the reduced number of animals available within individual herds only the correlations between daughter and dam, half sisters with a common sire and half sisters with a common dam have any meaning. These correlations are tabulated below for those herds which have 20 or more such pairs.

Table 12 shows that there is a fairly wide variation between the different herds in the amount of residual variation which exists within them. Some herds could change the form of their cattle by further selective breeding. The average residual heredity which remains out of the total which could have existed for the particular herd is rather small. This fact is further brought out in tables 13 and 14 for the two classes of half sisters.

TABLE 13

Correlation coefficients for the relation between the types of half sisters with a common sire.

HERD NO.	WEIGHT	HEIGHT AT WITHERS	DEPTH AT WITHERS	HEART GIRTH	PAUNCH GIRTH	WIDTH AT HIPS	BODY LENGTH	RUMP LENGTH	NO. OF PAIRS
19	0.02	0.00	−0.04	0.00	−0.03	0.02	0.01	0.09	960
27	0.13	−0.02	0.05	−0.03	−0.03	−0.07	−0.04	−0.01	418
47	0.01	−0.05	0.03	−0.06	−0.05	−0.03	−0.06	−0.04	514
52	−0.04	0.11	−0.02	−0.05	−0.05	0.01	0.07	−0.01	346
58	−0.08	−0.10	0.06	−0.03	−0.05	0.13	0.00	0.00	850
69	0.08	−0.01	−0.02	0.09	0.05	0.04	−0.04	0.09	486
77	0.00	0.18	0.02	0.01	0.06	0.10	0.14	0.01	3644
78	0.10	0.07	0.16	0.21	0.15	0.33	0.15	0.19	3610
88	−0.02	0.06	−0.06	0.00	0.03	0.11	0.24	0.03	768
102	0.09	0.06	0.12	0.11	0.03	0.09	0.08	0.29	1462
203	−0.03	−0.01	−0.01	−0.23	−0.05	0.14	−0.05	−0.03	484
Mean	0.02	0.02	0.02	0.00	0.01	0.08	0.05	0.06	

TABLE 14

Correlation coefficients for the relation between the types of half sisters with a common dam.

HERD NO.	WEIGHT	HEIGHT AT WITHERS	DEPTH AT WITHERS	HEART GIRTH	PAUNCH GIRTH	WIDTH AT HIPS	BODY LENGTH	RUMP LENGTH	NO. OF PAIRS
19	−0.23	0.13	−0.21	−0.33	−0.26	−0.42	0.05	−0.33	50
27	−0.13	0.38	0.02	0.47	−0.08	0.17	−0.42	−0.07	22
47	−0.22	−0.49	−0.07	−0.60	−0.17	−0.33	−0.20	−0.45	22
52	0.22	−0.36	−0.11	0.00	−0.35	−0.33	−0.17	−0.13	20
58	0.03	−0.25	−0.34	0.40	−0.20	0.15	−0.13	0.42	38
69	0.13	0.02	0.25	−0.06	0.23	−0.11	0.08	0.36	36
77	0.25	0.37	−0.11	0.47	0.37	0.51	0.24	0.29	44
78	0.31	0.19	0.18	0.19	0.09	0.24	0.10	0.30	116
88	0.07	−0.23	0.14	0.14	0.00	−0.09	−0.21	−0.33	34
102	−0.06	0.20	0.09	0.04	−0.01	−0.13	0.01	0.17	74
203	−0.39	−0.13	−0.57	−0.58	−0.55	−0.19	−0.03	−0.24	20
Mean	0.01	−0.02	0.06	0.01	−0.06	−0.05	−0.05	0.00	

The data of tables 12, 13 and 14 show that the Jersey herds have been differentiated into types which are quite pure in their inheritance. These types are different for different herds. The effect of the methods of breeding which have been employed have been to produce family groups which are rather homogeneous. To bring in genetic variability on which to work for further change would necessitate the interbreeding of some of these groups.

SUMMARY

This paper presents a summary of the body constitution of Jersey cattle as it is found after centuries of evolution under domestication and separation from other cattle for a period of nearly 200 years. The cattle utilized are pure-bred Jerseys owned within 15 states of the United States. Nearly 6000 relatively high-grade Jerseys form the basis for this study.

Data are presented to show the amount of residual inheritance for body constitution which remains within the breed as a whole and on the individual farms where the breeding is carried on. The parent and offspring and sibship correlations are presented. These correlations show that inheritance accounts for most of the variation in the size of these cattle, such environmental differences as do exist playing but little part in the ultimate constitution of these animals. The data show high coefficients of assortive mating. These are particularly noticeable for the hip width, body length and rump length. These coefficients are sufficiently large to indicate the separation of the Jersey cattle into non-interbreeding groups, each of these groups being characterized by a definite type. Study of the residual variation, subject to the control of the inheritance, which is left within the groups shows that it has been much reduced in the cattle of any one herd. The individual owners have consequently utilized to nearly its full measure the inheritance which was present initially within the breed. Further selection within the herds consequently must be devoted to the more refined points, or if extensive changes in type are desired resort must be had to crosses between families of the desired types.

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CATTLE INHERITANCE. I. COLOR[1]

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INTRODUCTION

A considerable amount of work has been done during the past fifteen years in the study of cattle inheritance. Each worker has taken up only a small portion, and no one has attempted to weld the whole mass into a compact whole. In this, and two other papers which are to follow, such an attempt will be made. The present paper discusses color inheritance, the second will deal with anatomical characters in general, and the third with milk production.

In most of the published papers the effects of each gene have not been carefully defined. This naturally has led to confusion. The following pages will contain as nearly as possible an exact description of the effects produced by each gene, together with its interactions, so far as they are known, with other genes. There probably is no truly logical order of gene presentation, but one sequence may lead to greater clarity than another. The one that seems most satisfactory will be followed here.

The present series of papers is not intended to be to any extent historical, nor will much space be devoted to a discussion of the relative merits of conflicting theories. The main purpose is to outline the present status as it appears to the author. In the course of this presentation hitherto unpublished facts will be given, and factors will be postulated and described which have not as yet appeared in print. The complete, supporting data will not be presented here, but will be published elsewhere when time permits.

COLOR GENES

In the course of the present paper postulated color genes occupying nineteen different loci will be described in detail. Some of those included are given with a certain degree of hesitancy due to the meager evidence in support of them. Others, not included among the above, but proposed by various authors, will be mentioned chiefly to demonstrate that they do not actually exist. Each of the genes will be designated by means of distinctive letters, in order to avoid confusion; but, to prevent duplication, the letters proposed by the author making the first description of the character will not always be used.

Red, R

So far as we know, red (*R*) is present in all cattle. It does not always show because there are so many other genes epistatic to it. We shall therefore have to assume for the present that it is always found in the homozygous condition, and consequently has no allelomorph. It causes the hairs to be red, and, when unaffected by other genes, is responsible for the brownish pigment in the skin of the nose and of the eyelids.

Black, B

The gene for black (*B*), found in Aberdeen-Angus and Holstein-Friesians, causes all the pigmented hairs on an animal to become black. Besides having this effect, it is also responsible for the black pigment in the skin, hoofs, tongue, lining of mouth, eyelids, nictitating membranes and "whites" of the eyes, provided the white spotting gene (*s*) does not interfere. The relationship between *B* and *s* will be taken up when the latter factor is discussed.

All that can be said concerning the allelomorph of black, *b*, is that it is the absence of *B*. Animals that are *bb* are not necessarily red, although, as will be shown directly, this represents in part the composition of a truly red animal.

Black Spotting, Bs

Black spotting (*Bs*) refers to the type of black found in Jerseys and Ayrshires, and presumably also in Brown Swiss. All the pigmented hairs are not black, and the black is concentrated in certain areas, forming variable sized spots with a somewhat indefinite outline. Since the black is not completely extended over the pigmented part of the coat, red, which is always present, has an opportunity to show. Even in the black areas the hairs may not be entirely black. Besides having the above mentioned effect on the hairs, the *Bs* gene, like the *B* gene, causes black pigment to appear in the skin, hoofs, nose, tongue, lining of mouth, eyelids and "whites" of eyes, if the animal is a self (*S*).

Another peculiarity of the *Bs* factor is that it is not fully expressed in the newly-born calf. Black pigment may be in the tongue and other parts above enumerated, but the black pigment in the hair creeps in gradually with increasing age, and males as a rule develop more black than the females. This sex-limited tendency will be discussed more fully under the factor pair *M* and *L*.

All that can be said concerning the recessive gene, *bs*, is that it is the absence of *Bs*. Since both *B* and *Bs* produce black hairs in one form or another, and since red (*R*) is always present, a *bb bsbs* would be red, with no black hairs showing, unless other genes exist which cause black pigment to appear in the hairs. A *bb bsbs* animal should in addition be devoid of black pigment in the skin, hoofs, nose, tongue, lining of mouth, eyelids and "whites" of the eyes, provided it does not carry the gene *Ps* for pigmented skin. This gene will be taken up later. The Guernsey breed is usually of the composition *bb bsbs*.

When Angus (*BB*) are crossed with Jerseys (*BsBs*) the offspring are black like the Angus, thus demonstrating that *B* is epistatic to *Bs*. Particulars concerning the F_2 of such a cross will be given after the factor for

brindling (*Br*) has been discussed. The interactions of *B* and *Bs* and their allelomorphs are as follows:

BB BsBs, black
BB bsbs, black
bb BsBs, black-and-red
bb bsbs, red

PEARL (1913) and HOOPER (1921) have postulated a separate dominant gene for black tongue color in Jerseys. The former states in a footnote that further studies indicate that tongue color depends upon two separate factors which show partial coupling on a 3:1:1:3 basis. Both base their conclusions on herd book records. Nevertheless, there seems to be no question but that in at least the great majority of the cases black tongue in Jerseys is due to the *Bs* factor, and not to a separate gene. HOOPER states that of the one thousand descriptions he examined in the registry books of the American Jersey Cattle Club there were only fifteen of self-colored animals that had light tongues and red switches. Such animals were probably *bb bs bs*. There is another more important cause for light-colored tongues in Jerseys and that is the white spotting gene (*s*) which brings about a lack of pigment in the tongue as well as in other parts of the body. The action of the *s* gene will be discussed more in detail later.

FUNKQUIST (1920) assumes that three pairs of factors govern the "muzzle" color of the Swedish breed of Stjernsund. The appearance of the pigmented hairs of this breed is not given, nor is any statement made concerning white spotting. Until one has more particulars concerning the whole animal it does not seem advisable to accept FUNKQUIST's three factor pair hypothesis.

Black Spotting Modifiers, *M* and *L*

In 1916 WENTWORTH reported on a sex-limited relation in the Ayrshire breed. He classified all individuals studied into either red-and-whites or black-and-whites and stated that heterozygotes were red-and-white if they were females and black-and-white if they were males. Such an explanation does not fit in with the factorial presentation given in this paper, and I shall endeavor to point out where it seems to be in error. In the first place there are black-and-white Ayrshires in Scotland that carry *B*, the factor causing all pigmented hairs to be black (KUHLMAN 1915b). There is no sex-limited effect here since all heterozygotes are black whether they are males or females.

The gene actually concerned is *Bs*, or black spotting. This factor is found in most Ayrshires and Jerseys, and, as before stated, the males in both breeds show more black than the females. If there were any sex-limited genes they would be modifiers of *Bs*. I have examined a fairly

large number of Ayrshires and Jerseys and find it impossible to separate them into two or three distinct classes, which should be possible if there were only one pair of factors affecting the amount of black that *Bs* produces in the hairs. There is a great deal of variation nevertheless and the males are on the average distinctly darker than the females. The sex-limited effect is present, but it seems to be due to more than one pair of modifiers. Since it is not known how many factors are concerned, it seems advisable to assume at least one pair for the time being. These allelomorphic genes are *M*, much black, and *L*, little black. *M* is dominant in the male and *L* in the female. A heterozygous male (*ML*), therefore, should show much black and a heterozygous female little black.

WENTWORTH described the sex-limited condition in Ayrshires only, but it applies equally as well to Jerseys. LUSH (1929) has recently written a paper, "Atavism in Jersey Cattle," which can be explained by the above interpretation. The case presented by him is as follows: A Jersey cow showing very much black (No. 47) was mated to a bull having very little black and produced a female offspring (No. 10), who from her description and photograph seems devoid of black, therefore being *bsbs*. The latter when mated to a bull showing much black produced a female (No. 34) as black as the granddam (No. 47), thus demonstrating atavism.

The following genetic interpretation is proposed:

♀ No. 47, *Bsbs MM* × ♂, *Bsbs LL* = ♀ No. 10, *bsbs ML*.

♀ No. 10, *bsbs ML* × ♂, *BsBs ML* (or *MM*) = ♀ No. 34, *Bsbs MM*.

M or *L* have no effect in the cow No. 10, since it is assumed that they act only in the presence of *Bs*.

There is another sex-limited effect in cattle which may possibly be due to the same modifiers that are responsible for the differences in the amount of black in Ayrshires and Jerseys. It is most noticeable in the red (*bb bsbs*) breeds, such as the Guernseys, Shorthorns and Herefords. In all of these breeds it is usual for the male gradually to attain a deeper shade of red than the females, as maturity is reached. The details concerning these changes have not as yet been investigated. If the same sex differences are present in the black breeds, such as the Aberdeen-Angus and the Holstein-Friesian, they are at least not so apparent.

Brindle, Br

Brindle usually does not occur in any of the pure breeds of cattle in America, but is found in scrubs, and has been reported among the F_2 generation of the Angus-Jersey cross and also among the F_1 generation of Jersey × Red Danish. There are a few cases known in purebred Ayrshires. The character itself consists of irregular, narrow stripes of black hair on a background of red.

Various attempts have been made to explain the inheritance of this character, but none are completely satisfactory. The most important contribution has been made by COLE (1925), who states that "it may be carried both in full blacks, such as Angus, or in clear reds, such as Red Danish, without being apparent. . . . It would appear to need a partial extension factor [*Bs*] to permit its expression; in the Red Danish such a factor is lacking, and in the Angus the brindling is masked by the action of a factor for the full extension of black [*B*]."

The explanation herein presented, which is essentially the same as COLE'S, is that the character, brindle, is due to the joint action of two genes, *Bs* and *Br*. The latter may be looked upon as a pattern factor which causes the black of a *Bs* animal to form into stripes. *Br* has no effect in a *B* animal, since *B* is epistatic to it, and none in a *bb bsbs* animal because there is no black present. Such an explanation fits in with the results obtained by PARLOUR (1913) and COLE (1924) in the F_2 of the Aberdeen Angus-Jersey cross. We would have to assume that many Aberdeen-Angus cattle carry the *Br* factor either in the heterozygous or the homozygous condition, and that many of them are homozygous for *bs*. For the sake of simplicity we shall make both breeds homozygous for all the genes concerned:

Angus (black) Jersey (black-and-red) (black)

$$BB\ bsbs\ BrBr \times bb\ BsBs\ brbr = Bb\ Bsbs\ Brbr$$

$$Bb\ Bsbs\ Brbr \times Bb\ Bsbs\ Brbr = \begin{cases} 27\ B\ Bs\ Br^2 \\ 9\ B\ Bs\ br \\ 9\ B\ bs\ Br \\ 3\ B\ bs\ br \end{cases} \text{48 blacks}$$

$$9\ b\ Bs\ Br, \quad \text{brindles}$$

$$3\ b\ Bs\ br, \quad \text{black-and-reds ("Jerseys")}$$

$$\begin{cases} 3\ b\ bs\ Br \\ 1\ b\ bs\ bs \end{cases} \text{4 reds ("Guernseys")}$$

Both investigators also describe in the F_2 a "very dark type of Jersey" (COLE 1924), and a black female "with fawn showing through" (PARLOUR 1913). The two descriptions seem to fit the same type of animal, and I shall attempt to explain it genetically when the factor *w* is discussed.

WRIEDT (1919) states that in the Telemark breed of Norway the only colors are brindle and red, and that brindle is dominant. To make these results agree with the factorial explanation given in this paper one would

[2] These represent the various factorial combinations in the F_2 generation. They are therefore zygotes, but the homozygous and heterozygous dominants are both represented by only one letter, as for example, *B*, and the recessives are also represented by means of only one letter (*b*, *bs*, and *br*).

have to assume that many of the reds in the Telemark breed carry *Bs*. WRIEDT gives no details concerning the appearance of these animals.

Intensity and Dilution, I, i; D, d

There seems to be good evidence that in cattle there is a form of dilution due to a dominant gene (*D*) and also another form due to a recessive gene (*i*). The former seems to be more pronounced in its effect, especially on black. A black animal carrying *D* is a dun. I have never seen such an animal and hence cannot from personal experience give a definite description of its appearance. It does not occur in any of the pure breeds of North America.

The recessive dilution factor (*i*) occurs in the dairy breeds, Jerseys and Guernseys, and is at least one of the causes for their being a lighter shade of red than such breeds as the Herefords, red Shorthorns and Ayrshires. There is no question but that there must be a number of genes responsible for the extreme variation in shade of red in the different breeds. The specific effect of each has not been determined, nor can one even say what is the precise effect produced by *i*.

If one examines the breeds carrying *B*, such as the Holsteins and Angus, he will find comparatively little variation in shade of black. It does not seem probable that this is because they are relatively homozygous for the dilution factors or their allelomorphs. The true explanation probably is that the dilution factors (except *D*) have less noticeable effect on black than they do on red. The best test for this hypothesis would be to determine the variation in shade of red in the self reds or the red-and-whites that are occasionally produced in the above black breeds.

The inheritance of dun has been studied in West Highland cattle by WATSON (1921). There is no question but that it is due to a dominant factor. However, WATSON is slightly sceptical of WRIGHT'S (1917) interpretation that it is the result of the dominant dilution factor (*D*) acting on black (*B*). He states that the results he has obtained can be explained equally as well by assuming that dun is due to a dominant color gene, and that when this gene is present the animal is a dun whether or not it carries black. Supplementary evidence strongly favors the interpretation given by WRIGHT. In the first place, yellows (dilute reds) as well as reds are found in the West Highland breed, which is what would be expected if there were a dominant dilution factor present. In the second place, red and black seem to be the only colors present in cattle, and chocolate seems to be the only other color found in mammals. Cattle would be unique if dun were present as a distinct color. WRIGHT (1917) is of the opinion that *D* is incompletely dominant to its allelomorph.

PITT (1920) has furnished somewhat meager evidence that *D*, or some

other factor having a similar effect, is present in the Hereford breed. She classifies her animals into "yellows" (light reds) and "clarets" (dark reds) and states that clarets tend to breed true, while yellows mated to clarets produce both yellows and clarets. She has no records of the matings of yellows to yellows because yellow was not a popular shade in the herd from which she obtained her data. The mating together of heterozygous yellows would have been the crucial test.

The results of POUND (1928) conflict somewhat with those of Miss PITT. He finds no true breeding shade of red, although he, like Miss PITT, did not mate yellows to yellows. His yellows seem to carry an incompletely dominant factor. If yellow Herefords actually carry *D* they should produce duns when mated to intense blacks.

I have been informed recently by Mr. E. N. WENTWORTH that many such crosses have been made on the ranges of the Southwest in the United States, and that all the offspring have been intense blacks, thus indicating the *D* is not carried by yellow Herefords.

Self and Recessive White Spotting, S, s

A self animal is entirely pigmented, and therefore shows no white spotting. The gene for self, *S*, is allelomorphic with *s*, the gene for recessive white spotting, but supposedly *S* is not completely dominant to *s* (COLE 1924). Part of this apparent incomplete dominance may be due to the fact that the ostensibly recessive white-spotted animal may also be carrying genes for dominant white spotting. The question will be discussed more in detail when the latter factors are being considered.

The gene *s* is quite variable in its expression due to modifying factors and also because of non-germinal variation. It seems reasonable to assume that if all variation in this character were germinal, the white spotting on the two sides of an animal, should be exactly the same. Since the two sides are generally unlike we would have to look upon the differences as being non-germinal in nature. DUNN, WEBB and SCHNEIDER (1923) have found a high correlation ($r = .97 \pm .002$) for the amount of black on the two sides of Holsteins. This, of course, does not take into consideration the distribution of the black and white areas.

One point concerning recessive white spotting which previously has not appeared in the literature is that *s* is responsible not only for white areas in the coat but also for unpigmented areas in the tongue, nose, lining of mouth, eyelids, nictitating membranes and "whites" of the eyes. This becomes apparent especially when the animals carry *B* or *Bs*. We have unpublished data (obtained by H. W. MATHEWS and R. R. OEHMCKE) showing that of the approximately one hundred animals making up the Holstein and Ayrshire herds at the KANSAS EXPERIMENT STATION all of them

have entirely unpigmented tongues, while the extent of the unpigmented areas in the other parts above enumerated is fairly closely correlated with the amount of white in the coat.

In making a study of spotting in Holsteins, LAUPRECHT (1926) has viewed it from the standpoint of the location of the pigmented areas rather than of the location of the white spotting. He postulates six paired pigment centers: 1. Eyes and cheeks; 2. Ears and neck; 3. Side of neck; 4. Upper arm and shoulders; 5. Side of body near last rib, and 6. Rump. He states that in certain cases the individual spot may be broken into several parts, or certain spots may be fused. The total amount of black is correlated with the amount at each center. If one examines a series of animals having progressively less pigment, he states that the pigment disappears first from the shoulders, then from the sides and finally from the rump, leaving pigmented areas only on the anterior end of the animal.

Since the factor *s* refers to white spotting it seems less difficult to view its action from the standpoint of the location of the white areas. I have not made a careful investigation of the matter, but my observations so far as they go tend to show that, in animals having only a small amount of white, the location of it is mostly on the limbs and the ventral side of the body. Animals having a large amount of white are white ventrally and posteriorly and to a less extent anteriorly. This last observation accords with that of LAUPRECHT.

Modifiers of *s*; *Lw* and *lw*, *Pl* and *pl*

Both LAUPRECHT (1926) and DUNN, WEBB and SCHNEIDER (1923) seem agreed, although they have expressed themselves somewhat differently, that a single pair of modifiers is to a large extent responsible for the varying amounts of white in Holsteins. These genes seem to have nothing to do with the pattern, but are entirely quantitative in their action. DUNN, WEBB and SCHNEIDER have furnished evidence that the factor causing a small amount of white (*Lw*) is incompletely dominant to its allelomorphs (*lw*). Hence animals carrying either a small amount of white or a large amount should breed true. LAUPRECHT maintains that such is the case in Holsteins. Confirmatory evidence may be obtained from other breeds in this country. Guernseys and Shorthorns almost invariably have a small amount of white spotting (*LwLw*) and seem to breed true without selection. Red-and-white Shorthorns, however, have even less white than do Guernseys, an indication that there may be other factors influencing the amount of white. On the other hand there are many Ayrshires that are almost entirely white (*lwlw*). I have looked over a fairly large number of matings in the KANSAS EXPERIMENT STATION Ayrshire herd, where both parents were largely white, and the offspring were similar to the

parents. Such animals tend to be unpigmented not only in the tongue but also on the nose, in the lining of the mouth, the eyelids, nictitating membranes and the "whites" of the eyes.

Another modifier of *s* has recently come to light (IBSEN and RIDDELL 1931). Animals carrying it, *Pl*, are pigmented to a large extent below the knees. The factor acts as an inhibitor of white in these regions. It also causes some irregularity of the white spotting on the head by adding small pigmented areas. As before indicated, the legs below the knees are not entirely pigmented, and the extent of the white seems to be somewhat correlated with the amount of white in other parts of the body. In "pigmented-legged" (*Pl*) animals the pigmentation seems to be concentrated around the hoofs and to extend upward, and is easily distinguished from cases where a pigmented spot spreads downward past the knee. The gene *Pl* seems to be found only in Ayrshires and Shorthorns in this country. Holsteins, Guernseys and other breeds are *plpl* and therefore one would expect them to be entirely white from the hoofs to the knees. There are a number of exceptions however. For example there are Holsteins with one or more legs entirely black. Oftentimes such animals have very little white and this may account for some of the cases. In others this is not a satisfactory explanation, and as yet no examination of the ancestry or of the progeny has been made to determine whether there is any evidence of the character being hereditary. Another exception occurs in Guernseys where a fairly large number of individuals are found with small pigmented patches directly above one or more hoofs. These patches are probably not due to the *Pl* factor, but whether or not they are inherited has not been determined.

FUNKQUIST and BOMAN (1923) postulate three pairs of genes affecting the amount of white on the heads of Holsteins and a red-and-white Swedish breed. In their paper they do not take into consideration the white spotting on other parts of the body. They postulate that an entirely pigmented head is due to the joint action of three recessive genes, and an entirely white one to at least two homozygous dominants plus either a homozygous or a heterozygous third dominant.

The facts, so far as I have been able to ascertain them in American Holsteins, are not in accord with this theory. Occasionally animals are found with heads almost entirely white even though the remainder of the body may be largely pigmented. One dominant gene is responsible for this condition, although the evidence as yet is not by any means complete. It is uncertain whether the gene is a modifier of *s* or is one for dominant white spotting.

Phenotypically, from the standpoint of the location of the white areas, a white-faced Holstein of the above description resembles typical speci-

mens of the Swiss Simmental breed. In Germany Simmentals are called "Fleckviehs," and LANG (1914) records a cross of this breed with a self-colored (*S*) breed known as the Limburger. The F_1 were pigmented except for their white faces, thus showing that the Fleckviehs carry a gene for dominant white spotting. It will be shown later that this gene is distinct from that producing the Hereford pattern. There is no record as yet of crosses between white-faced Holsteins and self-colored breeds.

Most Holsteins do not carry the dominant gene for white head, and yet there is much variation in the amount of white spotting in that region. There is no question in my mind but that for those animals lacking the dominant gene there is a close correlation between the amount of white on the body and the amount on the head. Animals of the composition *lwlw* have more white on the head than those that are either *Lwlw* or *LwLw*. This would be directly contrary to the theory of FUNKQUIST and BOMAN who postulate dominant genes for increased white spotting.

Occasionally pure white, or almost pure white, Ayrshires and Holsteins are found. Such animals are *ss lwlw* to begin with, and probably also carry "minus modifiers." The existence of the latter has not as yet been proved for this particular case. At any rate, animals that are practically white would have white heads which are due to the combined effect of several genes, some of which are known to be recessives.

HOOPER (1921) assumes there is a separate gene for white switch in Jerseys. He obtained his data from the registry books of the AMERICAN JERSEY CATTLE CLUB. An analysis of the data shows that the white switch was found mostly in animals that carried white spotting (*ss*) in other parts of the body. A few so-called selfs also had white switches. My observations lead me to believe that a white switch is only one of the manifestations of the *s* factor, and that, if an animal carries any white at all, the end of the tail is one of the places almost invariably affected. I have already called attention to the fact that the same applies in regard to the effect of *s* on the pigmentation of the tongue—animals that are *ss* almost invariably have unpigmented tongues.

Roan, N

The mode of inheritance of roan in Shorthorns and in crosses between Shorthorns and other breeds has attracted a great deal of attention in recent years. A number of different theories have been advanced, but it would require too much space to give all of them in detail.

The simplest, and probably the first, explanation given is that red and white are allelomorphic and that red roan is the heterozygote. One difficulty with this theory is that it cannot be applied satisfactorily to crosses between white Shorthorns and black breeds, such as the Angus. The F_1

from such a cross are "blue grays" and at first glance one might suppose that black also is allelomorphic with white. If such were the case the F_2 generation should consist only of whites, blue grays and blacks. There are actually two kinds of whites—those with red "points" and those with black, and, besides the blue grays and blacks already mentioned, red roans and reds. The results in the F_2 indicate that we are dealing with a dihybrid cross instead of a monohydrid. An apparently new and at the same time satisfactory explanation of the above results was devised, but it was found later that it had been antedated a number of years by CREW (1925). His theory as given in his own words (p. 68) is as follows: "All Shorthorns may be reds. If this is so, it is necessary only to postulate a series of modifying factors affecting (1) the extension or restriction and (2) the regional distribution of the colour to get roans and also parti-coloured beasts. A white would thus be duplex for the restriction, a roan one that is simplex. The factors affecting regional distribution [*s*] can act on a roan as well as on a solid colour."

CREW'S explanation is not quite as specific as the one I had in mind. In the latter the incompletely dominant factor *N* is postulated, which causes hair of any color to become devoid of pigment and therefore white. In the heterozygous condition (*Nn*) approximately one-half of the hairs interspersed throughout the pigmented areas are white, causing the animal to become a roan, while in homozygotes (*NN*) nearly all the hairs except some in the ears are white. Such an explanation can be readily applied to the white Shorthorn-Galloway cross reported by EVVARD, SHEARER, LINDSTROM and SMITH (1930), and seems more satisfactory than the one they themselves propose.

In their paper they put forward the following factor pairs to explain their results:

B = black.
b = red.
E = extension factor, causes pigment to cover whole coat.
e = absence of extension factor, causes pigment to be found only in points.
N = roan (sprinkling of white hairs) completely dominant to *n*.
n = no roan.

Using these factors, they designate a white Shorthorn as *bb ee NN* and a Galloway as *BB EE nn*.

Their theory seems quite inadequate even when applied to crosses within the Shorthorn breed. For example, if a white Shorthorn (*bb ee NN*) is mated to a red (*bb EE nn*), assuming both to be homozygous, the F_1's will be roans (*bb Ee Nn*), as would be expected, but in the F_2's there will be

many unusual genotypes. The F_2 phenotypic ratio should be nine roans: three reds: four whites, but of the roans one (*EE NN*) should be entirely homozygous, two (*EE Nn*) should produce no whites when inbred, two (*Ee NN*) should produce no reds when inbred, and the remaining four (*Ee Nn*) should breed like true roans and produce reds, whites and roans when inbred. Of the three reds in the F_2's, two (*Ee nn*) should produce whites when inbred, and one of the four whites (*ee nn*) should produce no roans when mated to reds.

If we use the genes suggested in this paper, remembering that my *N* is different from theirs and that a *bb* animal is also assumed to be *bsbs*, the crosses would be represented as follows:

$$\text{White } (bb\ NN) \times \text{red } (bb\ nn) = \text{Roan } (bb\ Nn)$$

$$bb\ Nn \times bb\ Nn = \begin{cases} bb\ NN \text{ (white)} \\ 2\ bb\ Nn \text{ (roan)} \\ bb\ nn \text{ (red)} \end{cases}$$

It is true that there are occasional exceptions. For example there are cases known where whites mated to reds produced reds instead of roans, but, if the interpretation of EVVARD and co-workers were correct, these would not be exceptions. They would be far too numerous to come under that classification. I shall attempt later to explain the exceptions.

Using the genes suggested above, the white Shorthorn-Galloway cross may be represented in the following manner:

White Shorthorn $(bb\ NN) \times$ Galloway $(BB\ nn) = Bb\ Nn$, blue gray[3]

$$Bb\ Nn \times Bb\ Nn = \begin{cases} 3B \begin{cases} NN, \text{ 3 whites, black points} \\ 2Nn, \text{ 6 blue grays} \\ nn, \text{ 3 blacks} \end{cases} \\ 1bb \begin{cases} NN, \text{ 1 white, red points} \\ 2Nn, \text{ 2 red roans} \\ nn, \text{ 1 red} \end{cases} \end{cases}$$

EVVARD and co-workers obtained the following offspring in the F_2 generation: 11 blue grays, 5 reds, 3 whites with black points, 1 black, and 1 red roan, or a total of 21. Their numbers are small. Using HARRIS's formula for goodness of fit and ELDERTON's table, for the interpretation proposed above, the probability (P) was found to be .0061.

In their F_2 generation EVVARD and co-workers obtained a number of animals that showed recessive white spotting (*s*). Since the P_1 Galloway

[3] The term "blue gray," employed by EVVARD and co-workers, represents conventional usage. From a factorial standpoint it would be more appropriate to designate such animals black roans, a term more easily contrasted with red roan.

cows and the F_1 blue grays were selfs (*S*), it would mean that the P_1 white Shorthorn bull must have been *ss*, which would not be apparent phenotypically because *N* in the homozygous condition is epistatic to *s*. The white Shorthorn bull thus would be *bb NN ss* and the black Galloway cows *BB nn SS*. Taking white spotting into consideration, they obtained 7 different phenotypes among their 21 F_2 animals. Theoretically there should have been 10 phenotypes. What seems rather remarkable is that when one determines P for their F_2 phenotypes, using again the factorial interpretation proposed in this paper, it is found to be .0371. The details have not been given due to lack of space.

Roan Modifier, *rm*

The main objection that EVVARD and co-workers have to other explanations than their own is that they do not explain exceptions. I have already pointed out that with their own explanation one would expect many more exceptions than actually occur. I have also mentioned that the chief exceptions are cases where white bulls, such as Whitehall Sultan, have produced some red offspring when mated to red cows. The figures for Whitehall Sultan as given by EVVARD and co-workers are 44 roans and 15 reds. Whitehall Sultan's son, Maxwalton Sultan, also white, produced 41 roans and 13 reds when mated to red cows.

SMITH (1925) reports the case of a bull that was a red roan when young which gradually lost his white hairs and became a red. This might possibly explain some of the reds produced in the white×red matings, but it undoubtedly would not explain all of them. One reason for this is that most purebreds are registered when young.

I am offering a tentative explanation for the above exceptions by postulating a new recessive modifying factor, *rm*. An animal of the composition *bb Nn rmrm* would by this hypothesis be red because *rm* (roan modifier) changes roan to red. A typical roan would be either *bb Nn RmRm* or *bb Nn Rmrm*. The mating of a white bull like Whitehall Sultan to red cows could be represented in various ways. White bulls would be either *RmRm*, *Rmrm*, or *rmrm* without any phenotypic effect, the one possible exception being that a *NN rmrm* animal might be a roan instead of a white. If this were the case it would be possible to produce a true-breeding strain of roans. Whitehall Sultan should be *Rmrm* at least, and the red cows that had red offspring by him should be either *Rmrm* or *rmrm*. Since the occurrence of *rm* in Shorthorns may be considered rather infrequent, most of the matings of a bull like Whitehall Sultan would be with *RmRm* red cows, and the offspring from such matings would be *bb Nn* (*RmRm* or *Rmrm*), or roans.

Although there is no definite proof of the existence of the factor *rm*, there are various ways in which a test could be made for it. The exceptional red cows produced in the white×red mating should be *bb Nn rmrm*. When mated to ordinary whites (*bb NN RmRm*) they should produce whites (*bb NN Rmrm*) as well as roans (*bb Nn Rmrm*), and when mated to ordinary reds (*bb nn RmRm*) they should produce roans (*bb Nn Rmrm*) as well as reds (*bb nn Rmrm*). It does not seem extravagant to propose a modifying factor like *rm* when one considers the great variation in the expression of the roan character in Shorthorns and the modifiers that are undoubtedly responsible for this variation.

One more point that probably should be made in regard to the interaction of *N* with other genes is that when a white Shorthorn (*bb bsbs NN*) is mated to a Jersey (*bb BsBs nn*) the offspring (*bb Bsbs Nn*) should be blue gray in spots and red roan on the remainder of the body if there is no white spotting present. I have seen animals that fill this description, but their parentage was unknown.

Recessive White, Wn

MANRESA, GONZALEZ, SARAO and ESGUERRA (1930) report on some crosses made in the Philippines between Native cattle and either Herefords or the Nellore breed of India, and also between Herefords and Nellores. Their description of the Nellore breed is as follows: "Nellores are phenotypically white, but indications are that they are really gray. White animals have unpigmented skins. The skins of Nellores are usually deeply pigmented, that is, almost black. For lack of a better term we will call the color of Nellores silver gray."

When Herefords were crossed with Nellores three types of offspring were produced, (1) brindled, (2) black, and (3) "dark fawn, with black extremities." The authors do not attempt to explain the inheritance of the Nellore's "silver gray" in the above cross, but do so in the cross between Native cattle and Nellores, where they obtained some "grays." They assume that "silver gray" is due to a dominant gene, designated by them *A*, which in the homozygous condition is completely epistatic to both the black and the dominant dilution factor (*D*) of the Native cattle, while it is incompletely epistatic when heterozygous.

It seems more logical to base the inheritance of "silver gray" on the results obtained when Nellores are crossed with Herefords, since we are better acquainted with the genetic make-up of the latter breed than of the Native cattle of the Philippines. If this is done, one is forced to conclude that the "silver gray" of the Nellores is actually a form of recessive white, the factor for which can be represented by *wn*, the *n* standing for Nellores, and thereby distinguishing this from other forms of recessive white which

may occur. The statement by MANRESA and co-workers that white animals have unpigmented skins is true, but their implication that they are no longer white if the skin is pigmented does not hold. White Silkies, a breed of domestic fowl, have pigmented skins and yet are considered to carry a factor for recessive white. It may also be (I have not had an opportunity to make a personal inspection) that the whites with black points produced in the F_2 of the White Shorthorn-Galloway cross have black skins. If the authors had said that true albinos do not have pigmented skins, it would not have been questioned.

The fact that Nellores when crossed with Herefords produce brindles, blacks and "fawns" demonstrates that this Indian breed carries *Bs* and sometimes *B*. The results from all three of their crosses agree with the assumption made by them that the brindling factor (*Br*) is carried by the Herefords, although there is still the possibility that it is also carried by the Nellores.

Several possible explanations may be given for the black skin of the Nellores. One is that it is due to a separate factor. Another is that *wn* is incompletely epistatic to either *Bs* or *B*. Both of these factors cause the production of black in the skin as well as in the hair and other parts of the body, and the *wn* gene when homozygous may prevent the formation of pigmentation in the hair, but have no effect on the skin.

Information received recently from Mr. E. N. WENTWORTH bears out the above interpretation. Shortly after the War he made a microscopic examination of white hairs from several Nellore cattle exhibited at the Fort Worth, Texas, Show. Even macroscopically the hairs did not seem a pure white, and this was borne out under the microscope by the fact that black and red pigment granules were found sparsely scattered throughout the hair. Based on the above observations, it seems logical to interpret the *wn* gene as one causing extreme dilution. The presence of both black and red pigment granules in the hairs indicates that the animals investigated were *bb*, but at the same time carried *Bs*. It also seems reasonable to assume that since Nellores carry either *B* or *Bs*, or both, that the *wn* gene causes extreme dilution in the hairs, but has relatively less effect on the pigment of the skin, which consequently remains black.

I have no explanation to offer for the "grays" obtained in many cases when Nellores are crossed with Native cattle unless it be that the Native cattle are often *Wnwn*, thus offering opportunity for the outcropping of "silver grays" (*wnwn*) in the offspring.

The foregoing account possibly may be made a little clearer by giving the assumed composition of the Herefords and Nellores, making them heterozygous for certain factors in order to show all the different types of offspring produced:

Herefords Nellores

$$bb\ bsbs\ Brbr\ WnWn \times Bb\ BsBs\ brbr\ wnwn =$$

$$\begin{cases} Bb\ Bsbs \begin{cases} Brbr\ Wnwn, \text{ black} \\ brbr\ Wnwn, \text{ black} \end{cases} \\ bb\ Bsbs \begin{cases} Brbr\ Wnwn, \text{ brindle} \\ brbr\ Wnwn, \text{ "fawn"} \end{cases} \end{cases}$$

KUIPER (1921) makes the statement that a recessive form of white is found in both the Pembroke and the Highland breeds of cattle, but he furnishes no references or proofs. So far as I have been able to determine, the evidence thus far is quite inconclusive. WALLACE (1907) states that the white Pembrokes are "throw backs" from the black breed of South Wales, which would make them appear to be recessive whites, but he also states that when a black bull of the South Wales breed is mated to white Pembroke cows "a large proportion of his progeny take after the mothers in being white with black points." The last statement would best fit in with the assumption that many of the white cows are heterozygous dominants. There is still the possibility, however, that most of the South Wales black bulls are heterozygous for recessive white.

WILSON (1909) has made a genetic analysis of color in the Highland breed. He is of the opinion that white cattle imported by the Romans into England from southern Europe were one of the ancestors of this breed, and that for many years occasional whites occurred amongst them. The fact that no whites appear at present may more easily be explained by assuming that the ancestral white was due to a dominant rather than to a recessive gene.

Dominant White, Wp

It has long been known that the white English Park cattle (Cadzow Castle Park, et cetera) carry a factor for dominant white. WRIEDT (1929) has called attention to this fact recently. He states that the same factor is found in Swedish mountain cattle. Animals carrying the gene are not completely white, but, in addition to having pigmented ears, may have colored flecks on the body. SANDERS (1925) has a photograph of six Cadzow Castle Park cattle. One of these animals is to a large extent hidden by the others, and a few are partially hidden. All six, however, seem to have black "points," four have small pigmented spots on the neck, and five have pigmented areas on the legs which correspond in appearance to those produced by the *Pl* (pigmented leg) factor in Ayrshires.

It would seem from the above that the white of English Park cattle actually is a form of dominant white spotting which causes the animal to

be largely white. I have called the factor producing it *Wp*, the *p* having reference to Park. As yet I have not come across any reference to its genetic relations with self (*S*). There are photographs, each showing non-whites in a group of White Park cattle, and these non-whites have all had the appearance of being self blacks. In every case they are said to be offspring of white parents, and, if this is true, it would mean that black (*B*) and self (*S*) are common in these animals and that *Wp* is incompletely epistatic to self. A *wpwp* animal would merely be one that did not carry dominant white (*Wp*).

Dominant White Spotting

Hereford Pattern, S^H, *S*,*s*

The typical modern Hereford is red with a white head, white feet and tip of tail, white stripe of varying width and length over and parallel to the backbone in the region of the shoulders, and with white on the entire ventral side of the body. It has been customary to assume that the inheritance is very simple, *H* generally being used to designate the factor for the white spotting and *h* its absence. No discussion that I have seen has ever considered the relation of the above factor *H* to either *S* or *s*. When one takes this relationship into consideration, there are two main possibilities as to the part which *H* plays in the production of the whole Hereford coat pattern, under which heading would be included the pigmented as well as the white areas.

One possibility is that *H* is responsible for both the white and the pigmented areas and their distribution, and is a dominant allelomorph of *S* and *s*. Instead of being allelomorphic, it could be epistatic to these factors; but this will not be considered for the time being. The above explanation would account for the apparent ease with which the Hereford pattern has been fixed, since it would be necessary to have the animal homozygous for only one factor. On the other hand it would not explain the genetic composition of the "Fleckvieh" (Simmental) breed of Germany, which in addition to a white face and white extremities, also has white in places found only on a *ss* animal (LANG 1914). There is no evidence available, however, to show that the dominant factor which produces white face in Fleckviehs is the same factor that is responsible for white face in Herefords.

If we assume an allelomorphic relationship between the three factors it could be represented as follows, the factors being given in their order of dominance:

S^H = Hereford pattern.
S = self.
s = recessive white spotting.

There is evidence that S^H is incompletely dominant to S and completely dominant to s. This is shown by the fact that when Herefords are crossed with either Angus or self Jerseys the offspring have little or no white on the legs and less white on other parts of the body than the Hereford parent. When they are crossed with white-spotted (*ss*) breeds like the Holsteins the offspring show no diminution of white.

Some evidence obtained recently from the herd of Mr. JOHN SHOEBROOK of Atchison County, Kansas, fits in well with the allelomorphic conception. He crossed purebred Herefords (S^HS^H) with high grade white spotted Shorthorns (*ss*), mated the crossbreds to Herefords, and continued backcrossing to Herefords for a number of generations. Recently he took one of the Hereford-marked bulls that he had produced as a result of the above sort of breeding and mated him to similar cows. Most of the offspring had typical Hereford markings. There were only two exceptions, and both were intermediate white-spotted individuals (*ss Lwlw*). From the results obtained, on the multiple allelomorphic interpretation, it would mean that the bull and at least two of the cows are S^Hs.

The other possibility in regard to the genetics of the typical Hereford pattern is that the factor H is responsible only for the white spotting and that S causes the remainder of the coat to be pigmented. In such a case H would be epistatic to S, and a true breeding Hereford would have to be homozygous for two factors, H and S. This sort of explanation could be applied satisfactorily to the Fleckvieh breed. The latter would be of the composition *HH ss*, because of the excessive white spotting appearing on the body. One drawback to a two factor hypothesis for the Hereford coat is that one would expect greater difficulty to have been experienced in fixing it than was actually the case. Even at the present time it should not have been unusual for sporadic cases of self reds, white-faced animals showing recessive white spotting, and others showing only recessive white spotting, to appear.

Of the two explanations proposed I am inclined to favor the multiple allelomorphic one. There is also a third possibility, previously mentioned, that of a factor similar in its effect to S^H but which is epistatic rather than allelomorphic to S and s. All three possibilities could be tested by appropriate matings. What seems to be the most feasible method is to mate Herefords to *ss* animals, preferably some homozygous for *lw*, and to backcross the hybrids to *ss lwlw* individuals. If S^H is allelomorphic to S and s, there will be only two phenotypes resulting from the backcross—"Herefords" and recessive white spotted animals. If it is epistatic, there should be in addition to the above two phenotypes, an occasional self (S). If the Hereford pattern is due to the combined effects of H and S, the backcross should produce four different phenotypes, with equal numbers of each:

$HHSS \times hhss = HhSs$

$HhSs \times hhss =$
- $HhSs$, Hereford pattern.
- $Hhss$, Hereford pattern, plus recessive white spotting.
- $hhSs$, self.
- $hhss$, recessive white spotting.

It should be evident that if either the multiple allelomorphic or the epistatic explanation proves to be the correct one, it will be necessary to postulate a different dominant factor for the white face of the Fleckvieh breed. I believe it will be found that the gene causing the white face of the Fleckviehs is different from that producing the white face of the Herefords.

S^H Modifiers, *Lw*, *lw*; *Rn*, *rn*; *Re*, *re*.

Although we are uncertain as to the true genetic explanation for the Hereford pattern, there is no question but that so far as we know every individual in the breed is homozygous for the gene, or genes, producing this pattern. At the same time there is a great deal of variation in its expression, due to modifying factors. Some interesting, though meager, results have been obtained on this point by Miss PITT (1920).

She looks upon the factors she proposes as allelomorphs to one producing the Hereford pattern, but furnishes no proofs. It seems more appropriate to consider them modifiers of S^H, and as such they will be discussed. The first is a recessive factor which causes the animals to have an increased amount of white. From her standpoint it is a special factor found in Herefords, but there is some, as yet unpublished, evidence accumulated which justifies one in assuming, at least tentatively, that her factor and *lw* are identical. The evidence above mentioned has been obtained from the crossing of Herefords with Holstein nurse cows. The two Hereford bulls used had slightly more white than would be considered desirable, and, judging by their offspring as well as by their appearance, were of the composition *Lwlw*. The cows were either *Lwlw* or *lwlw*. The five calves produced thus far have either had an extremely large amount of white (*lwlw*) or a slight excess (*Lwlw*). No *Lw Lw* animals have appeared, but the total number as yet is small. Observations on purebred Hereford matings also indicate the incomplete dominance mentioned above. Miss PITT implies that the dominance is complete. For the present, at least, it seems advisable to assume that *Lw* and *lw* are modifiers of the Hereford pattern factor (S^H) as well as of that for recessive white spotting (*s*).

Rn, *rn*

Miss PITT proposes another modifying factor which causes the entire

dorsal surface of the neck and shoulders to be pigmented. An animal with such a character she describes as "dark-necked," while the Hereford breeders of the United States refer to it as "red-necked." The latter term seems the more appropriate of the two. The factor, which I have designated *Rn*, is incompletely dominant to *rn*, the absence of red neck. Thus, a *Rn rn* animal, according to Miss PITT, would have a small amount of white dorsally on the neck, and if at the same time it were *lwlw*, the white in that region would be increased so appreciably that it would look like an individual with the normal amount.

Since it has been assumed that *Lw* and its allelomorph are modifiers of both *s* and S^H, it seems desirable to speculate on the possibility of *Rn* being also a modifier of *s*. Some evidence on that point has been obtained from Hereford-Shorthorn crosses. Shorthorn breeders for many years have made an effort to decrease the amount of white spotting in their breed by careful selection. The effect has been quite marked, and as a result Shorthorns have much less white than Guernseys, which as a breed are *LwLw*. Many of the Hereford-Shorthorn crossbreds have red necks and in general an increase in pigmentation, seemingly due to the Shorthorn parent. The evidence, such as it is, fits in with the hypothesis that the Shorthorn breed not only is *LwLw*, but in addition carries another dominant modifier of *s*, in this case assumed to be *Rn*.

Re, re

A third modifier proposed by Miss PITT is responsible for the production of red hair around each eye. Most Herefords have entirely white faces and as a result the eyes of many of them become injured if they are long exposed to the glare of the sun. Those with "red eyes" are less liable to such injury. The factor producing red eye, *Re*, is considered to be completely dominant to *re*, absence of red eye, although Miss PITT has no definite information on this point. She also has little or no evidence that *rere* animals breed true. POUND (1932), on the other hand, makes the statement that "animals with white at the eye, when bred together, produced practically nothing but calves like the parents." He makes the additional implication that *Re*, instead of always causing red hair to form about the eye, may sometimes produce only some pigmentation in the skin of the eyelids. It seems impossible at present even to guess what effect *Re* would have on a *ss* animal. So far as I have been able to determine, "red eye" is found only in Herefords or in other breeds carrying the S^H gene.

There are many variations in the Hereford pattern and the three pairs of modifiers proposed by Miss PITT would not by any means account for all of them. Moreover, she does not assume that they do. POUND (1932)

has called attention to one of the more striking variations not discussed by Miss PITT. It is known amongst breeders as "lineback." Animals with this characteristic are white along the top line, from the head to, or almost to, the tail. Oftentimes it is associated with reduced pigmentation over the entire body, but there are many exceptions to such a rule. It is not known as yet whether or not lineback is due to a separate modifier, but, the fact that two animals lacking the character may produce an offspring that shows it, makes it look as if it were inherited as a recessive. Very few lineback bulls are retained for breeding because of the undesirability of the character.

Amongst early Herefords there were many individuals with "brockle" faces. Such animals had large blotches of pigmented hair on an otherwise white face. At the present time similarly marked animals are found on those ranches of the western United States where there has been some intercrossing between Herefords and Shorthorns. A more careful examination of the pictures of early Herefords and also of the living descendants of crossbreds, shows that all brockle-faced cattle are "pigmented-legged" (*Pl*). There seems to be no question but that the same factor produces both effects. Since the brockle face is due to a dominant gene, no difficulty should have been experienced in eliminating it by selection from the Hereford breed. The *Pl* factor is quite common amongst Shorthorns, and thus furnishes the explanation for the presence of brockle faces amongst the descendants of the Hereford-Shorthorn crossbreds. Such animals are raised only for the market and no selection is practiced in regard to their pattern.

Although it is not intended in the present paper to discuss all of the European breeds, I cannot resist the temptation to take into consideration one that apparently carries the S^H gene. This is the Groningen (called "Groninger" by KUIPER [1921]) breed of Holland.

The observations on Groningens are based on eight photographs obtained from three different sources. All eight animals (three males and five females) have white faces but are pigmented around the eyes (*Re*). These pigmented spots are continuous with the pigment of the neck. The pigment on the head and neck extends further forward than it does in typical Herefords. None of the eight animals have dorsal white stripes. This makes them red-necked (*Rn*). The three bulls apparently have no white on the belly, while all five cows have white udders and an additional small amount of white ventrally on the body (*LwLw*). In some of the photographs the hoofs, and that part of the legs directly above them, do not show distinctly, making it difficult to determine the amount of white in that region. Nevertheless, four animals are white on all four legs. It extends upward only a short distance, however. One bull unmistakably has four entirely

pigmented legs, and a cow has white on both rear legs and none on the forelegs. The location of the white on the legs of the other two animals cannot be determined with certainty.

It is assumed that the eight Groningens discussed represent desirable specimens of the breed. All of them presumably carry two completely dominant (*Re* and *Rn*) and one incompletely dominant (*Lw*) modifier of S^H. Each of these modifiers tends to increase the amount of pigmentation on the animal. If careful selection for increased pigmentation has been practiced, one would expect most of the breed to be *LwLw* because of the incomplete dominance of *Lw* to *lw;* but there should still be a number of individuals heterozygous for either of the other two modifiers, who, when mated, would produce some offspring either with a clear white face (*rere*) or with a white stripe dorsally on the neck (*rnrn*). Critical evidence on the S^H interpretation could also be obtained. If S^H actually is responsible for the Groningen pattern, no white-faced animals showing recessive white spotting (*s*) should be produced.

The Norman breed of France also apparently carries the S^H gene. I have at my disposal at present the photograph of only one individual, and hence will attempt no generalizations. This animal, a bull, is pigmented-legged and brockle-faced (*Pl*), has pigment around the eyes (*Re*) and a dorsal white stripe on the neck (*rn*). It has enough white on the belly to be classified as a *Lwlw* individual. One striking characteristic that differentiates it from Herefords and Groningens is the marked irregularity of outline of the ventral edges of the pigmented area. Similar irregularities of outline are found in Ayrshires and Guernseys, and the evidence from these breeds, based on general observations, is that the character is due to one or more recessive genes.

Dominant White Spotting

Dutch Belt, S^D, *S*, *s*

All the published evidence on the inheritance of Dutch belting seems to have been furnished by KUIPER (1920, 1921). The photographs and diagrams in his papers show that the belt, which is white, varies in width and regularity of outline and extends around the body back of the shoulders and forelegs. The character is found chiefly in the breed known in this country as the Dutch Belted Cattle, and in Holland as Lakenvelders. The breed seems to have originated in Holland, and its distribution is not widespread. The typical color pattern is black and white with the white being almost entirely restricted to the belt. The most common exception seems to be a small amount of white directly above the rear hoofs.

KUIPER (1921) states that the off-types in the breed are self blacks, red dutch belted, and self reds, and that these are due to the occasional out-

crossing that has been practiced. It is significant that he does not mention the occurrence of animals which have the typical recessive white spotting (*s*) in addition to the dutch belt, nor of animals having only the recessive white spotting. If the first of these two types never occurs it will have an important bearing on the mode of inheritance of the belted condition.

The explanation as given by KUIPER is that there are two pairs of factors concerned. These are the factor for self (*S*) and the dominant factor for white belt (he uses *B* for belt and *b* for absence of belt). His factor *B* is considered to be epistatic to self. At first glance this seems to be a satisfactory explanation, but, as previously intimated, there is at least one serious objection, which is that, since the breed is to some extent heterozygous, there should be some matings in which both parents are *Ss*, and who as a result should produce an occasional *ss* animal having in addition a dutch belt. One gets the impression from KUIPER that animals of this description are never produced.

If this is the case, it seems advisable to fall back on a multiple allelomorphic explanation, as was done with the factor for Hereford pattern (S^H). One would, under the circumstances, have to assume that a single factor not only produces the white belt but is also responsible for the remainder of the coat being pigmented. The gene having this effect, S^D, like S^H, would be allelomorphic to *S* and *s*, and would be dominant to them. With the factors in an allelomorphic series, it would be impossible for a dutch belted animal to show any recessive white spotting unless S^D happened to be incompletely dominant to *s*. The results recorded in KUIPER'S paper show that the dominance is complete. The multiple allelomorphic interpretation would also explain the relative infrequency of recessive white-spotted (*ss*) animals in the Dutch Belted breed. It would be necessary for both parents to be S^Ds before any could be produced. If one parent were S^DS and the other S^Ds, the off-types would be selfs.

The chief data presented by KUIPER are the results obtained from the mating of a purebred Dutch Belted bull with fifty-three *ss* cows, and with one "Groninger Zwartblaard" (S^H), cow. Only the F_1 animals were reported on. The bull, according to the interpretation herein presented, was of the composition S^DS. There were fifty-five F_1 animals. Of these, twenty-seven were belted, "twenty-four or twenty-five" were self-colored, and "three or four" were "pied." Some of the selfs had small white spots. The pied individuals were assumed by KUIPER to be of the composition *ss*. The location of the white spotting on two of the latter is shown by means of diagrams, and there is no question in my mind but that they are imperfectly marked dutch belted individuals. They have less white than is found normally in the Dutch Belted breed, but the same amount of white in a *ss* animal would be located almost entirely on the belly and the feet,

and would not extend up so far along the sides. I shall venture the guess that their *ss* mothers showed very little white spotting.

KUIPER offers the tentative explanation that the bull was *B*(belt)*bSs*, and that these factors are linked with approximately 12.5 percent crossing over. There are two flaws in his explanation. One is, as already pointed out, that there are no *ss* animals in the F_1 generation, and the other is that he assumes a *Bbss* animal to be as typically dutch belted as one of the composition *BbSs*. The multiple allelomorphic representation of the cross would be:

$$S^DS \times ss = \begin{cases} S^Ds, \text{ dutch belted.} \\ Ss, \text{ self.} \end{cases}$$

If one considers the "pied" individuals as dutch belted, the numbers obtained were approximately thirty dutch belted and twenty-five selfs. A critical test of the correctness of the two theories would be the mating of the F_1 dutch belted to *ss* animals.

Since S^D and S^H are assumed to be allelomorphic, it would be of interest to determine the interaction of the two genes in a heterozygote (S^DS^H). So far as I know only one such individual has been reported on thus far. It has already been referred to in the detailed account of KUIPER'S data. The purebred Dutch Belted (S^D) bull was mated to a Groningen cow, belonging to a breed presumably carrying the S^H gene. The one offspring, as represented by KUIPER in a very small and somewhat indistinct diagram, has the white face of the Groningen mother and the belt of the father. Both hind legs also seem to be white. The two allelomorphic genes affect different parts of the body and both are enabled to express themselves in the heterozygote.

Dominant White Spotting

Colorsided, S^C, *S*, *s*

The term "colorsided" was probably originated by WRIEDT (1925). The character is found in a number of European breeds of cattle, and it is also found amongst yaks and zebus. In MCCANDLISH'S paper (1920), dealing with crosses between purebreds and scrubs, there is a photograph of an Arkansas scrub which is colorsided, and also one of her colorsided offspring by a purebred Holstein bull.

The term is to a large extent self-explanatory. Colorsided animals are pigmented on both sides, with the remainder of the body white. Thus, they have a fairly wide white stripe along the entire backbone, and the belly and brisket are white. There is also some white on the head and legs. The line of demarkation between the pigmented areas and the white is very irregular, and some mottling is generally found in the white areas on the head.

The data on the inheritance of colorsided have been collected by WRIEDT (1925). A number of different matings are reported, but the numbers are comparatively small. One exception is the mating of Telemarks and Troenders, colorsided Norwegian breeds, with selfs, such as the Aberdeen-Angus. There were eighteen offspring altogether, and all of them had white bellies and short, narrow, white spinal stripes, but had no white whatever on the heads and necks. WRIEDT concludes that colorsided is incompletely dominant to self.

Colorsided animals were mated to two different recessive white-spotted (*ss*) breeds. The two individuals used in the first, a red-and-white Swedish breed, were males and had only a small amount of white, which was restricted to the belly. The numbers here, also, are fairly large. Fourteen offspring altogether were produced. All had less white than the colorsided parents. WRIEDT concludes that colorsided is incompletely dominant to the white spotting of the Swedish breed. I am inclined to think that the apparent incomplete dominance is due to the plus modifiers (*Lw*, et cetera) carried by that breed. This becomes more apparent when the next cross is considered.

The other breed with recessive white spotting was the Holstein-Friesian. It was crossed with Telemarks and the four offspring were found to have the typical colorsided pattern of the Telemarks. One of the F_1 cows was backcrossed to a Holstein-Friesian bull and produced three daughters. One was colorsided and the other two were typically Holstein in regard to color markings. The colorsided daughter was mated to another Holstein bull and produced a colorsided female offspring, which when mated to a Holstein bull also had a colorsided offspring. WRIEDT concludes that colorsided is completely dominant to the recessive white spotting of Holsteins. None of the Holsteins were described in regard to the amount of white they carried. The chances are, however, that they had more white than the two Swedish red-and-whites described above.

If WRIEDT's statements are taken literally it would mean that, since he considers colorsided dominant to both self and recessive white spotting, he also considers the three factors to be allelomorphic. He does not use any letter to designate the factor for colorsided, thus making it difficult to determine his exact ideas on the subject. His evidence, however, seems to fit in best with the multiple allelomorphic explanation. The alternative explanation would be that colorsided is due to the combined action of two factors, that for self (*S*), and a white spotting factor, epistatic to self, which is responsible for the white found in a colorsided animal. If larger numbers had been obtained in the backcross of F_1 with Holsteins, the question could have been settled with more certainty. In such a cross, if the two factor theory were correct, selfs should have been produced in addition to

the colorsided and Holsteins. Also, on such a theory, one would expect some colorsided animals to occur which showed recessive white spotting. These should be found occasionally also in the colorsided purebreds, since one would not expect all the individuals in the breed to be homozygous for the two factors mentioned above. There is nothing in WRIEDT's paper, however, to indicate that they are ever produced.

Adopting the multiple allelomorphic explanation, it seems advisable to use the symbol S^C for the colorsided factor. Like its allelomorphs S^H and S^D, it is responsible not only for the white but for the pigmented part of the coat. Based on the information supplied by WRIEDT, S^C is incompletely dominant to S and completely dominant to s. There seems to be evidence that the modifying factors which change the expression of s have corresponding effects on S^C. It will be of interest to obtain the phenotypic interactions of S^H, S^D and S^C. One is probably justified in predicting that any heterozygote will show the effects of the two factors that are present.

Dominant White Spotting

Inguinal White, *In*

The evidence furnished by GOWEN (1918) is fairly convincing that the factor which is responsible for inguinal white spotting is not allelomorphic with S and s. If it were, it would not only produce the small white spot in the inguinal region (on either the teats or the udders of females and on either the rudimentary teats or the sheath and scrotum of males), but would also cause the remainder of the body to be pigmented. There are animals, especially in the Aberdeen-Angus breed, which are entirely pigmented except for the inguinal white spot, but there are also Holsteins which when mated to selfs produce offspring with inguinal white spotting, thus demonstrating that *ss* animals may carry the inguinal white factor.

GOWEN did not discuss the possibility of the factor for inguinal white being part of an allelomorphic series. He stated that the character is due to a dominant factor. *In* is suggested here as the symbol for this factor. Its allelomorph, *in*, is merely the absence of the dominant gene.

Inguinal white can be demonstrated best in animals carrying the factor for self, in this way proving that *In* is epistatic to *S*. Animals that are *ss* usually are white along the entire underline, making it impossible to determine by inspection whether or not *In* is present. The presence of *In* in an *ss* animal can be shown only by a breeding test:

White Spotted *Self* *Inguinal White*

$$ss\ InIn \times SS\ inin = Ss\ Inin$$

Such a cross would on the face of it appear to be an example of the in-

complete dominance of *S* to *s*. This possibility was referred to earlier when the last named allelomorphs were discussed.

The data presented by GOWEN are the following. The mating together of heterozygous inguinal spotted animals produced 21 with the spot and 6 without. Heterozygous animals mated to recessives had 10 offspring with the spot and 8 without. GOWEN feels somewhat hesitant about fully accepting the factor. It should be quite a simple matter to test its validity by making careful observations within the Aberdeen-Angus breed where recessive white spotting is not present to obscure results.

Whitening, w

The Jersey breed color is very often described as "fawn." On closer inspection one finds that this color is made up of several components, one of which has hitherto not been described. Besides carrying the black spotting factor, *Bs*, and some form of dilution (either *i* or *D*), there is also apparently another factor which produces the effect here given the name of "whitening." The most striking manifestation of this gene is the whitish muzzle which surrounds the usually black nose. The factor, which is recessive and is designated *w*, also causes some of the hairs on the inside of the ear to be white or whitish, and is responsible for many white or whitish hairs on the belly, udder and inside of rear legs. The amount of whitening varies considerably. Often it extends over the entire body, but always to a less degree dorsally than ventrally. In extreme cases the apparent diluting effect brought about by the factor is marked. Animals with extreme whitening, which at the same time carry much black; due to *Bs* and modifiers, are grayish in appearance.

No formal experiments have been conducted to determine the mode of inheritance of whitening. The evidence given here is not absolute, but it all points to the conclusion that the character is due to a recessive factor. Its dominant allelomorph, *W*, prevents the appearance of the character.

Some of the supporting evidence will be mentioned. In the first place, all Jerseys seem to be homozygous for *w* even though no selection apparently has been practiced. Secondly, when Jerseys are crossed with Angus, Holsteins or Herefords, the whitening does not show in the F_1 generation, but does reappear in the F_2.

There is a possibility that *w*, in the homozygous condition, may have an effect in the presence of *B*. PARLOUR (1913), describing an F_2 animal from a Jersey-Angus cross, states that it is black "with fawn showing through." COLE (1924), describing an F_2 animal from a similar cross, calls it a "very dark type of Jersey." I have seen a purebred Jersey cow that was practically self black except for the whitish hairs on the muzzle. She was mated to a typically colored purebred Jersey bull and produced a daughter simi-

lar to herself. It seems reasonable to assume that mother and daughter, and also the animals mentioned by PARLOUR and COLE, carry *B* instead of the *Bs* factor usually found in Jerseys. If they do carry *B*, it means that *w* has less effect in the presence of *B* than it has in the presence of *Bs*. A well-informed Jersey breeder has made the statement that many of the "Majesty" line of purebred Jerseys are as black as the mother and daughter described above.

COLE (1924), commenting on the F_2 generation of the Jersey-Angus cross, states, "In no case so far, however, has the typical light fawn of the Jersey been exactly reproduced." The reason for this becomes apparent when one realizes that a typical Jersey should be homozygous for at least four recessive genes. Such a statement is true especially if the Angus P_1 carries the factor for brindling (*Br*). The typical Jersey would be of the composition *bb Bs*(*Bs* or *bs*) *brbr ii ww*. The dominant dilution factor, *D*, does not enter into the above cross since the black of the F_1 is practically as intense as that of the Angus P_1.

Pigmented (Black) Skin Spotting, Ps

Both the typical Guernsey and the typical Shorthorn are of the composition *bb bsbs*. If one examines the noses of individuals in either of these breeds he will find that they are usually yellowish-brown in color, the intensity of the color of the skin of the nose being correlated to a large extent with the shade of the hair. Since Shorthorns have hair of a deeper red than Guernseys, they also have noses that are more brownish in color. White Shorthorns have as darkly pigmented noses as the reds. The reason for this is that they carry the same modifiers of intensity as the reds, but the presence of these modifiers is not so apparent, due to the epistatic effect of *N* in the homozygous condition. This pigmented condition of the nose is not the result of a separate factor, but is merely the effect of the interaction of red (*R*) with factors normally present in the two breeds.

The noses of all Guernseys and Shorthorns, however, are not entirely yellowish-brown. Many have black spots of varying sizes interspersed through the brown, and sometimes the entire nose is black. In Guernseys one also finds black hairs on the muzzle, which surrounds the nose. The black hairs on the muzzle, however, are not necessarily associated with black spots on the nose. I have not seen any Shorthorns with black hairs on the muzzle. This may be due to the small number which have been examined for that purpose.

In other breeds, like the Holsteins and Ayrshires, the nose may occasionally be entirely devoid of pigment because of the action of the *s* factor in combination with its modifier, *lw*. Noses devoid of pigment would be pinkish in appearance since the blood has an opportunity to show through.

Holsteins and Ayrshires carry either *B* or *Bs*. Both of these factors cause black pigment to form in the nose. Both breeds are *ss*. The combination of either *B* or *Bs* with *s* generally results in an animal with black as well as white spots on the nose. Such black spotting is different from that, described above, which occurs in Guernseys and Shorthorns, although there are indications that the Guernsey type of black spotting also is found in Holsteins and Ayrshires.

Up to the present there have been no published reports on the inheritance of "smutty" (black-spotted) noses in Guernseys and Shorthorns, but there is a published statement by Miss PITT (1920) on the inheritance of the character in Herefords. She asserts that when pigmented nose occurs in the breed, the pigmented spots may be brown alone, black alone, but most frequently they are both black and brown. She does not try to account for the brown spotting. Since Herefords are *bb bsbs* like Guernseys and Shorthorns, it seems reasonable to assume that they would be brown-nosed also were it not for the S^H factor which usually is responsible for the head being devoid of pigment and therefore white. In certain cases, as shown by Miss PITT, some of the brown shows on the nose in spite of the S^H factor. It is not known whether or not this is due to a modifier of S^H.

There seems to be no question but that there is an inherited tendency for black pigment to form in the skin even though the hair above is white. I have seen the character in both Holsteins and Ayrshires. It is easier to detect it in these breeds because there are so many individuals with a large amount of white spotting. Guernseys and Shorthorns have very little white hair, and it is much more difficult to determine the skin color under pigmented hair. The best place, other than the nose, to look for either black or brown skin spots in Guernseys and Shorthorns is the udder and the teats. Oftentimes these spots are found in both the inguinal region and the nose, but occasionally they are found in only one or the other location.

Miss PITT (1920) has postulated a dominant factor for pigmented nose. Her data are very meager—the mating of heterozygotes with recessives resulted in three with pigmented noses and three without. A purported homozygous dominant mated to recessives produced four, all of which had pigment in the nose. She advances no evidence to show that when both parents have clean noses they always breed true.

Under the circumstances it seems more reasonable to suggest a dominant factor *Ps*, for black pigmented skin spots, which may fall almost anywhere on the body, but which would be most noticeable on the bare skin. With such a factor it would be possible for animals to carry it and still have clean noses. It would be very difficult to rid a breed of a factor of this kind if one took the nose only into consideration in determining the presence of the factor. Animals of the composition *psps*, on the above interpretation,

would have no black skin spots whatever, at least none that are due to *Ps*.

There is some evidence, far from complete, which indicates that the *Ps* factor expresses itself gradually in the growing animal. Up to the present it is based entirely on the apparent fact that young animals are more free from the character than older ones.

GENETIC COMPOSITION OF VARIOUS BREEDS

The foregoing account includes the description of a large number of characters for which the mode of inheritance is fairly well established. It also includes a few about which there remains some question. It is now proposed to take all of the above genes into consideration in presenting the factorial composition in regard to color for a number of the best known breeds of cattle in the United States. At the same time other characters in each breed will be mentioned whose inheritance has either not as yet been determined, or has not been fully enough investigated. This last list will not by any means be complete.

It will be impossible to take into consideration all the genes described, in giving the factorial composition of each breed, since each breed has not been tested for all of the genes. The method that will be followed is to present the composition as based upon the appearance of the most common type in the breed. If there are any phenotypic variations, the genes for these are placed directly under those representing the common type. The genotypic variations will not be indicated.

Sometimes it is difficult to determine what are the normal characteristics of a breed. For example, there are no data available concerning the frequency of inguinal white (*In*) in either the Angus or the white-spotted (*s*) breeds. I am assuming that this character is usually lacking. Since red (*R*) is assumed always to be present, it will save space by not including it in the factorial composition. Where there are modifying factors these will be placed directly after the genes they modify. The presence of some factors, which have no phenotypic effect in certain combinations, has been determined by means of breeding tests. These genes will be mentioned in the discussion after the genetic formula for the breed has been given.

Aberdeen-Angus

With the above considerations in mind, the genetic color formula for a typical Aberdeen-Angus may be represented as follows:

$$BB\ dd\ \frac{II\ inin}{In in}\ nn\ SS\ WW\ WnWn\ wpwp.$$

There is some variation in shade of black in the Angus breed. Such variation may be due to the fact that a few animals are *ii*, but there is no

evidence on the subject. Besides, much remains to be learned concerning the genetic cause for most of the variation in intensity in all of the breeds.

Some information concerning hypostatic genes carried by the Angus breed may be obtained when one takes into consideration the red calves that are occasionally produced by heterozygous blacks (*Bb*). COLE and JONES (1920) do not give a detailed description of such animals, but one gets the impression from their paper that the reds they have seen are of a uniformly deep shade and therefore do not carry the gene for black spotting, *Bs*. WALLACE (1907) has photographs of two red Angus which give every indication of being of the composition *bsbs*. All the evidence available fits in with the assumption that the Angus breed as a whole is homozygous for *bs*. On the other hand, evidence from the F_2 of Jersey-Angus crosses (COLE 1925) demonstrates that individuals of the Angus breed may carry the gene for brindling, *Br*.

Certain other variations have been observed. TEMPLETON (1923) reports on a white-faced red calf, produced when a *Bb* Angus bull was mated to a Hereford cow, that had "a dark muzzle and a dark tongue." Presumably the muzzle and tongue carried black pigment, and the hair did not. The black pigment probably would not be due to *Ps* since it was not in spots. There seems to be a possibility here of a hitherto unreported gene that causes all of the skin to carry black pigment even though the hair is red or white.

At the International Livestock Exposition of 1931, held at Chicago, Professor A. D. WEBER of the KANSAS STATE COLLEGE took special note of the red Angus shown, and has kindly given me permission to make use of the information he obtained. One animal is of especial interest. It was a purebred red steer and had the typical brownish nose and eyelids, but in addition had distinctly black hairs on the face and the poll. If it had carried the *Bs* gene the nose and eyelids would have been black. A possibility, pointed out by Mr. E. N. WENTWORTH, is that the apparently black hairs actually may be very intense red. He has examined under the microscope "black" hairs from red Shorthorns and has found them to be made up of very closely packed red granules.

One other point might be mentioned, and that is in connection with the presence or absence of the gene for pigmented leg (*Pl*) in the Angus breed. Invariably, so far as I have been able to ascertain, an animal, usually black, with a clean white face is produced when a purebred Angus is mated to a purebred Hereford. All purebred Herefords are *plpl*, and the crossbred sometimes would be "brockle"-faced if *Pl* were ever carried by the Angus parent. Thus we are justified in concluding that at least most Angus are of the composition *plpl*.

Holstein-Friesian

From a color gene standpoint Holsteins differ from the Angus breed chiefly in that they carry recessive white spotting, *s*, while the Angus are *SS*. Since the Holsteins are *ss* it makes it possible to determine what modifiers of *s* they carry, but makes it impossible to ascertain by inspection whether or not they carry *In*. The genetic composition of typical Holsteins therefore would be:

BB dd II nn ps ps ss Lwlw plpl WW WnWn wpwp
Psps lwlw
LwLw

It is assumed that animals with an intermediate amount of white spotting (*Lwlw*) are most common and that those with a very small amount (*LwLw*) are least common.

DETLEFSEN (1920) reports a peculiar case, from the standpoint of inheritance, in Holsteins, the information concerning which was supplied by the owner of the herd in which it occurred. Two albinos (even the eyes were devoid of pigment, and therefore pink) of opposite sex were produced from normally pigmented parents. These albinos when mated produced four albino offspring. From the evidence at this point the albinism would seem to be due to a recessive gene. However, when the albino male was mated to grade Holstein cows, all of the twenty offspring were albinos, the male thus appearing to be a homozygous dominant. It seems possible that the owner was not so anxious to get at the facts as to prove the "prepotency" of his albino herd, for that reason making the albinism due to a dominant as well as to a recessive gene when it probably was due to only the latter.

An animal described by LAUPRECHT (1928) possibly carries a new gene. It is a tricolor produced by normally pigmented Holsteins, and is unique in that the black spots are sharply differentiated from the red. In the colored plate it looks distinctly different from a *Bs* animal, such as an Ayrshire, where the black tends to blend with the red. One gets the impression that the animal carries *B*, but that this is only partially extended as in tortoiseshell guinea pigs. The cow showing the above character unfortunately proved to be sterile.

Four common variations might be referred to at this time. One is the tendency, previously mentioned in this paper, for animals to be produced with one or more legs entirely pigmented. It is not known whether or not this has an hereditary basis. Another variation is the "Ayrshire" type of spotting. In most Holsteins the white spotting is so distributed that the black areas are in big blotches, while in Ayrshires the pigmented areas form small spots. I have seen a calf, produced by an Ayrshire cow mated

to a Holstein bull, which had typical Holstein color markings. Such a result would be in line with the interpretation that Holsteins in general carry a dominant modifier of *s*, while the Ayrshires carry the recessive allelomorph. Additional data could be obtained within the Holstein and the Ayrshire breeds as well as by breed crosses.

A third variation also has to do with white spotting. I am referring to animals having heads almost entirely white even though the remainder of the body may be to a large extent pigmented. As previously stated, either a dominant modifier of *s* or a dominant gene for white spotting is responsible.

The last variation is evident only in calves. Animals possessing it have hair which fades to a reddish slate color previous to shedding. In other calves of the same age there will be no such change, the hair remaining black throughout. The character is probably inherited, but the data available are very incomplete.

Jersey

The chief difference between a Jersey and either an Angus or a Holstein is that the two latter carry *B* while most Jerseys are *bb*. Jerseys also are distinctive in that they are *ww*. The formula for typical animals of the breed is:

bb brbr BsBsMM L dd ii nn SS ww WnWn wpwp
Bb LL Dd ss LwLw
bsbs

The above formula should convey to the reader a number of different points, all of which have previously been made in the body of the paper. Two might be mentioned here. In the first place the extremely black animals with white muzzles, occasionally found, are assumed to be *Bb*, and, in the second place, those with flesh colored noses, *bb bsbs*.

Some explanation is necessary in regard to the placing of *dd* as the most usual composition of Jerseys and of *Dd* as being of rarer occurrence. In the previous discussion of *D* and *i* it was stated that *D* had a more pronounced diluting effect than *i*. Black animals carrying *D* are duns. However, most Jersey-Angus crossbreds are intense blacks, thus indicating that the Jersey parents were *dd*. KUHLMAN (1915a) states that duns are sometimes produced when the above cross is made. One would have to assume that the Jersey parents of these animals carried *D*. It is not known with certainty whether a Jersey carrying *D* can be distinguished from a *dd* animal, but, since *D* has a more pronounced effect than *i*, one can be reasonably certain that cream colored animals have a greater probability of carrying *D* than those of a darker shade.

Ayrshire

Ayrshires differ from Jerseys in two main particulars. They are white-spotted, as contrasted with self, and are intense rather than dilute. Their composition is:

bb brbr BsBs ML dd II nn psps ss lwlw plpl WW WnWn wpwp
Bb Brbr LL Psps Lwlw Plpl
MM LwLw
bsbs

The formula shows that there are a number of alternative conditions in the breed. The vast majority, if not all, of the animals in this country are *bb*, but there are some in Scotland that carry *B*. A few in this country show brindling (*Br*), and there are also a few that have flesh colored noses and are at the same time without any black pigment in the hair (*bsbs*). Some show black pigment in skin which is under white hair (*Ps*), while a larger proportion have pigmented areas of hair above the hoofs and below the knees (*Pl*).

There are a number of peculiarities in regard to white spotting in Ayrshires which may be due to modifiers of *s*. One has already been pointed out in the discussion of Holsteins. I refer to the tendency for the pigmented spots to be small and therefore comparatively numerous. If one examines a number of animals he will find considerable variation in the degree to which this breaking up into small pigmented areas has been carried on. An apparently accompanying phenomenon is the irregularity in outline of both large and small spots.

Another type of small spots entirely different from the above also occurs. That already described seems to be due to the breaking up of a large pigmented area into numerous smaller ones. The type about to be described occurs in white areas and consists of small pigmented flecks. This kind of spotting would tend to increase the amount of pigment carried by the animal while the other would tend to decrease it.

Still another peculiarity in regard to white spotting in Ayrshires is the presence of "halos." These surround the pigmented spots and consist of either black or red hairs, or both, that are intermingled with the white, and extend out only a short distance.

Hereford

The genetic color formula of the typical Hereford is rather long because of the special modifiers of the Hereford pattern:

bb bsbs dd II nn psps $S^{H}S^{H}$ rnrn rere LwLw plpl WW WnWn wpwp
Psps Rnrn Rere Lwlw
Bs? lwlw

Miss PITT (1920) believes that yellow Herefords carry a dominant dilution factor, but there seems to be no question that it is not *D*. Evidence from crosses shows that many Herefords carry the brindling gene, *Br*. This, however, is not apparent phenotypically in purebred animals. The fact that some animals have what seem to be black hairs in spots fits in with the supposition that *Bs* may sometimes be carried. Further evidence is necessary before the question can be settled. As pointed out previously, more data are necessary in order to work out the inheritance of "lineback," apparently due to a recessive gene.

Shorthorn

The most characteristic color gene in Shorthorns is *N*, which is responsible for roans (*Nn*) and whites (*NN*). The breed is also noteworthy in that, although no animal seems to carry the gene for self (*S*), a large majority of the *nn* individuals show very little white spotting. Some of the white spotting on roan animals apparently is due to *N* and its modifiers. Shorthorn breeders have made a conscious effort to reduce the amount of white spotting in their animals. They were not aware of the genes involved, but they have unconsciously retained *Pl* in order to keep pigment on the legs and head, they have decreased the amount of white by making most of their animals *LwLw*, and, finally, they have kept a third dominant modifier (tentatively assumed to be *Rn*) which made possible a further reduction. The formula for the breed is:

bb bsbs dd II Nn RmRm psps ss LwLw RnRn? PlPl WW WnWn wpwp
rmrm Psps Lwlw rnrn pl pl
nn lwlw
NN

There are probably many unsettled questions in regard to color inheritance in Shorthorns. Only two will be mentioned here. In the first place, there seems to be no doubt but that there are a number of modifiers affecting the expression of roan. One, *rm*, has already been postulated in this paper. The others, judging by the kind of variations found in the Shorthorn breed, apparently control the relative numbers of white and pigmented hairs, and still others control the uniformity with which this relationship extends over the entire body. There is also possibly an age effect, some modifiers tending to increase the relative numbers of white hairs with increasing age, and others having the opposite effect.

The second problem can be stated more briefly. I am referring to the extreme intensity of red found in the breed. Shorthorns are a deeper red than any other of the breeds considered in this study, and there is undoubtedly a factorial basis for this difference.

Guernsey

The typical Guernsey has the following color formula:

bb bsbs dd ii nn psps ss LwLw pl pl WW WnWn wpwp
Dd? *Psps* *Lwlw*
lwlw

There is probably some question as to whether or not the Guernsey breed ever carries dominant dilution (*D*). It depends partially on whether the "brownish-black" obtained by GOWEN (1918) in an Angus-Guernsey crossbred was a true dun. There is the possibility that the Angus parent was *Ii* and that the crossbred was a recessive dilute (*ii*).

Many Guernseys have hair which is much more dilute on the muzzle and on the inner sides of the rear legs than on the remainder of the body. It is not improbable that this is due to the same gene (*w*) that causes whitening in Jerseys. If it is, we would have to assume that the effect is less pronounced in Guernseys than in Jerseys, possibly due to the absence of the *Bs* gene in the former breed. The matter could be tested by an examination of *bsbs* purebred Jerseys or by crossing a Jersey with a light muzzled Guernsey.

Occasionally what appear to be black hairs are found interspersed with the red on various parts of the body of purebred Guernseys. The area covered is comparatively small, as in the red Angus previously described, and usually consists of the muzzle or the tail. The skin of the nose of such animals may or may not carry black pigment. If these effects are due to the *Bs* gene, one would have to assume that they have been greatly reduced because of the selection of minus modifiers.

It has been mentioned previously that a fairly large proportion of Guernseys have small pigmented areas of hair directly above one or more of the hoofs. The pigmented leg gene, *Pl*, does not seem to be the cause for these spots, nor has it been ascertained that any particular gene is responsible. There is the possibility that such spots may occur in *ss* animals that are at the same time *LwLw*, and who therefore have a comparatively small amount of white spotting. If such is the case, *LwLw* Holsteins should also show this type of spotting. Further investigation is necessary.

Two peculiarities that Guernseys have in common with Ayrshires might be mentioned. One is the "halo," the chief difference between the two breeds being that it consists only of red hairs in the Guernsey, while both black and red hairs may be found in it in the Ayrshire, due to the *Bs* gene. The other is the irregularity of outline of the pigmented areas in certain animals. In Ayrshires nearly all individuals have this irregularity of outline, but in Guernseys it is less common. It should therefore be less difficult

to get crucial data on the mode of inheritance in the latter breed since both types of spotting are available for study.

There are several other breeds in the United States that have a fairly large number of representatives, as for instance the Brown Swiss, but I have not had enough personal experience with them to make a critical discussion. The Guernseys, therefore, will be the last breed to be considered.

SOMATIC COLOR MUTATIONS

Several color variations have been reported which without much doubt are somatic mutations. They have not been described as such in the original papers, but they will be given that classification here.

Two cases are from LAUPRECHT (1928), one from HORLACHER (1928), and the fourth is from unpublished material. One of the animals described by LAUPRECHT was a "Schlesischen Landvieh." Individuals in this breed are normally red-and-white. The exceptional animal had in addition to the red-and-white a large patch of black on the left shoulder. From a genetic standpoint one would have to suppose that the animal was *bb*, but that a mutation had occurred in one or more of the somatic cells, and persisted in their descendants, changing a *b* to *B*.

HORLACHER's case is similar to the above, and the explanation would be the same. Here a black spot (17 cm×8 cm) enclosed by red was found on the left side of the neck of a purebred Hereford. HORLACHER states that black spots such as that described are occasionally found in both purebred Herefords and Shorthorns.

LAUPRECHT's second case was found in a Holstein-Friesian herd in Holland. The animal was a black-and-white with a small red spot about 5 cm across. Red-and-white Holsteins are fairly common in Holland and the females can be registered. Red-and-white males, however, cannot. In the United States no red-and-white Holsteins can be registered. One would therefore expect that a large proportion of black-and-whites in Holland are *Bb*. If the animal described by LAUPRECHT were of this composition the red spot could be explained by assuming that the *B* mutated to *b* in possibly one somatic cell, and that the spot was produced by the descendants of this cell.

The last case to be described was found amongst a large number of Hereford steers on the ranch of Mr. DAN CASEMENT of Manhattan, Kansas. The animal in question was not a purebred, but nevertheless both parents had the typical Hereford color and pattern. The somatic mutation (or mutations) was responsible for the production of two distinct roan areas on the posterior part of the animal. One of these areas, circular in shape and at least one foot in diameter, was on the left thigh, while the other, consisting of two spots, was on the inner sides of both hind legs.

Mr. CASEMENT informed me that amongst his Herefords he has seen black spots similar to the one reported by HORLACHER on one in every three hundred or so individuals, but that he has never before seen an animal with roan areas. Herefords are *nn* in composition. The somatic mutation consisted in the changing of an *n* to *N* in possibly two somewhat separated somatic cells.

Mr. E. N. WENTWORTH states that when the skin of either red or black-haired animals is blistered as a result of the application of concentrated dips or crude oil, or if the branding operation has been incompletely performed, there will subsequently be a sprinkling of white hairs in those areas, thereby producing a roan effect. Mr. CASEMENT's roan-spotted Hereford, however, has not received any of the above types of treatment.

SUMMARY

In summarizing the results of a paper of this kind it would be a tedious and at the same time an unprofitable task to attempt to delimit the new material in it. It should be sufficient to state that a complete revision of color inheritance concepts in cattle has been aimed at, and that during this process many changes have been advocated and much new matter added. In presenting the latter it has been deemed inexpedient to give the supporting evidence in detail.

One gene, that for red (*R*), is assumed to be always homozygous. Seventeen factor pairs and an allelomorphic series made up of five genes are described in detail and their interactions, as far as they are known, given.

The factorial composition, from the standpoint of color, is given for the seven leading cattle breeds of the United States. Attention is also called to a number of other color characters in these breeds of which the mode of inheritance has not as yet been sufficiently determined. Several somatic color mutations are described and an attempt is made to explain their occurrence.

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